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American Veterinary Medical Association

FORMERLY
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(Original Official Organ U. S. Vet. Med. Ass'n.)

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H. Preston Hoskins, Secretary-Editor, 1230 W. Washington Blvd., Chicago, Ill.

Vol. LXXXI, N. S. Vol. 34 **July, 1932** **No. 1**

CONGRATULATIONS, IDAHO

On June 1, Idaho was officially designated as a modified accredited area. This is the first state west of the Mississippi River to be placed on the honor roll, and the seventh to become accredited, the others being Maine, North Carolina, Michigan, Indiana, Wisconsin and Ohio.

Coöperative tuberculosis eradication began in Idaho in 1919, on an individual-herd basis, but in 1922 the state and federal governments took up the work on the county-wide or so-called area plan of testing, in coöperation with county officials and cattle-owners. Since the work began, nearly 900,000 cattle have received the tuberculin test, resulting in the disclosure of about 4,500 reactors, which were removed and slaughtered.

Gooding County was the first county in the State to become a modified accredited area, in June, 1924. This was followed, in August, 1925, by placing Minnidoka County in the modified accredited area. Then, in April, 1926, Gem and Lemhi counties were added to the list. The remaining 40 counties were added from time to time. During the last 18 months, many range cattle were tested under the provisions which exempt a large portion of the cattle from being tested in cases where no reactors are found among those tested. The cattle-owners of Idaho are to be

congratulated on this accomplishment, which would not have been possible but for the splendid coöperation given the county, state and federal officials.

TWO NEW EXECUTIVE BOARD MEMBERS

Elections for members of the Executive Board in two districts came to a close on June 23. The official canvass of the ballots showed an unusually close race in each election.

In District 1 Dr. A. E. Cameron, of Ottawa, nosed out Dr. Seymour Hadwen, of Toronto, by the narrow margin of three votes. In District 9 Dr. Harry W. Jakeman, of Boston, Mass., was declared a winner by a margin of fifteen votes over the runner-up. District 9 consists of the six New England states and New York. The vote in the Empire State was divided among three candidates. The New England vote was split between two.

The members-elect will take office at the close of the meeting in Atlanta. Dr. Cameron succeeds Dr. George Hilton, who this year will close fourteen years as a member of the Executive Board. Dr. Jakeman, the first member of the Board from the New England states, succeeds Dr. D. H. Udall, who filled the short term in District 9, following the redistricting which took place in 1930.

DR. H. W. JAKEMAN
Member-elect, Executive Board, District 9.

NEW DEAN AT CORNELL

The New York State Veterinary College at Cornell University has a new dean in the person of Dr. William Arthur Hagan, whose election was ratified by the Board of Trustees on June 20. The institution had been without a dean since the death of Dr. Pierre A. Fish, February 19, 1931. In the interim, a committee consisting of Drs. Earl Sunderville, R. R. Birch and W. A. Hagan administered the affairs of the N. Y. State Veterinary College.

Dr. Hagan takes his new post well prepared for the heavy responsibilities that go with such a position. Although not yet thirty-nine years of age, he has had both training and experience that admirably equip him to continue the work so well started

DR. W. A. HAGAN

by the late Dr. Veranus A. Moore and continued for a short time by the late Dr. Pierre A. Fish.

Following his graduation from the Kansas State College, in 1915, Dr. Hagan did graduate work at Cornell University and received the Master of Science degree in 1917. After teaching a year at Kansas State College, Dr. Hagan returned to Cornell as an instructor. He rapidly rose to the rank of professor and head of the Department of Pathology and Bacteriology. In 1921-22, while on leave of absence, he studied at the Rockefeller Institute for Medical Research, at Princeton, N. J., and in 1925-26, he continued his graduate studies at the Robert Koch Institute for Infectious Diseases, in Berlin, Germany, as an Exchange Fellow of the International Education Board.

Dr. Hagan has always taken an active interest in the A. V. M. A. At present he is chairman of the Section on Research, with the three previous years to his credit as secretary of the Section. He recently finished a three-year term as A. V. M. A. Representative to the National Research Council.

In expressing our confidence in Doctor Hagan, and in congratulating Cornell upon the selection of an outstanding veterinarian and scientist to direct her veterinary college, it is appropriate to repeat a remark made by a prominent member of the veterinary profession a few years ago. He said: "Doctor Hagan has a tremendous amount of energy and an unusual capacity for work." This is reflected in the large number of scientific articles which have appeared under his name during recent years.

Congratulations, Cornell! Best wishes, Dean Hagan!

MEMBERS VISIT NEW OFFICES

During the first two months of our occupancy of the new headquarters in Chicago, nine members of the A. V. M. A. called on various missions. The first was Dr. E. L. Quitman, of Chicago, in fact, a near neighbor, as his office is only a few blocks away. Dr. L. A. Merillat was also among the early visitors, as was Dr. E. A. Cahill, who had just recently returned from a three-month business trip in South America. Drs. Merillat and Cahill were members of the committee which selected the location for the Chicago offices.

The first out-of-town visitor was Dr. J. P. Bushong, of Los Angeles, California, who was on his way east to attend a meeting in Washington, D. C. Dr. E. M. Nighbert, of Washington, called during our absence from the city. Dr. R. A. Runnells, of Ames, Iowa, stopped long enough to go over a number of matters in connection with a manuscript he had submitted for publication in the JOURNAL. Dr. L. A. Mosher, of Atlanta, Georgia, chairman of the Committee on Local Arrangements, took advantage of the opportunity afforded by a trip north to call at the office and confer with the Secretary concerning a number of details in connection with the convention.

Dr. Geo. W. Cober, of Oklahoma City, Okla., temporarily located in Chicago, paid his respects on June 18. Several days later, Dr. N. S. Mayo, of Highland Park, Ill., dropped in for a conference on several matters concerning the activities of the Committee on Education. Dr. Wm. C. Prouse, of Minneapolis,

Minn., passed through Chicago twice in May, en route to and from Philadelphia, and called the office by phone both times. Close train connections in the Windy City prevented him from calling at the office.

The next time we have occasion to mention visitors we hope the number will be larger. The latch-string is always out.

DR. E. L. QUITMAN
First A. V. M. A. member to visit Chicago office.

Bird's-eye view of the Atlanta program

	Monday Aug. 22	Tuesday Aug. 23	Wednesday Aug. 24	Thursday Aug. 25	Friday Aug. 26
Morning	Meetings of Committees	Opening Session	Sectional Meetings	Sectional Meetings	Clinic
Afternoon	Meeting of Executive Board	General Session	General Session	General Session	Clinic
Evening	Southern States Vet. Med. Association Meeting	Alumni Meetings — President's Reception	Banquet	General Session — Papers and Entertainment	

APPLICATIONS FOR MEMBERSHIP

The economic dislocation has had its effect on the number of applications for membership received during the past year. This is not surprising, when we realize that practically no line of endeavor has escaped. Every individual and every organization has suffered to some degree. Few of our resident secretaries have felt that the time was opportune for putting on membership campaigns, although this has been done in several instances where there were indications that such efforts would bear fruit. More than the usual effort has been put forth to keep members in the ranks, on the theory that a member saved is a member gained. Veterinarians have had their incomes reduced, along with everybody else, and naturally there has been a tendency to curtail expenses by relinquishing memberships in organizations. From observations that we have made, it would appear that the A. V. M. A. has fared better in this repsect than many other organizations.

Every six months we remind our members of the modus operandi for joining the A. V. M. A. We find that this saves us much correspondence. The rules are simple. Here they are:

> Applications for membership shall be made upon blanks furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Applications must be accompanied by a membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Applications must be filed with the Secretary and examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of the applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BARNES, CHARLES F. c/o Interstate Packing Co., Winona, Minn.
D. V. M., Grand Rapids Veterinary College, 1915
Vouchers: John L. Myers and C. M. Heth.

BECHTOL, LAUREN L. Okolona, Ohio
D. V. M., Ohio State University, 1932
Vouchers: W. F. Guard and W. R. Krill.

COON, ELWYN W. Forest Grove, Ore.
D. V. M., Iowa State College, 1932
Vouchers: C. H. Covault, C. H. Stange and W. B. Coon.

DAVIS, DOUGLAS L. 254 Capitol Place, Atlanta, Ga.
D. V. M., University of Georgia, 1926
Vouchers: L. A. Mosher and J. E. Severin.

GADD, JOHN D. 512 S. 42nd St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1932
Vouchers: M. A. Emmerson and G. A. Dick.

GRINSTEAD, EMIL E. 207 E. 3rd St., Cle Elum, Wash.
B. S., D. V. M., State College of Washington, 1932
Vouchers: G. W. McNutt and E. E. Wegner.

HEAGERTY, ALOYSIUS K. 5211 Windsor Mill Road, Baltimore, Md.
D. V. S., United States College of Veterinary Surgeons, 1910
Vouchers: E. M. Pickens and R. C. Reed.

KNAPP, JOHN H. Ohio State University, Columbus, Ohio
D. V. M., Ohio State University, 1932
Vouchers: W. F. Guard and W. R. Krill.

LADSON, THOMAS A. Olney, Md.
D. V. S., United States College of Veterinary Surgeons, 1905
Vouchers: E. M. Pickens and Alexander Gow, Jr.

LOCKWOOD, CHARLES H. 905 New Jersey Ave. N. W., Washington, D. C.
D. V. S., National Veterinary College, 1896
Vouchers: H. E. Moskey and L. T. Giltner.

MOYER, RAYMOND S. 609 Quebec St., Allentown, Pa.
V. M. D., University of Pennsylvania, 1932
Vouchers: M. A. Emmerson and G. A. Dick.

NORLING-CHRISTENSEN, OLOF 254 Olentangy St., Columbus, Ohio
D. V. M., Ohio State University, 1932
Vouchers: W. F. Guard and W. R. Krill.

ROHRER, ARTHUR A. 1852 N. Fourth St., Columbus, Ohio
D. V. M., Ohio State University, 1932
Vouchers: W. F. Guard and W. R. Krill.

STARR, G. H. Box 56, National Stock Yards, Ill.
D. V. M., McKillip Veterinary College, 1917
Vouchers: A. C. Walls and C. F. Payne.

STERNFELS, MARK 560 W. 180th St., New York, N. Y.
D. V. M., Cornell University, 1932
Vouchers: E. Sunderville and D. H. Udall.

WASTRACK, WILLIAM R. Cedarburg, Wis.
B. V. Sc., Ontario Veterinary College, 1925
Vouchers: James S. Healy and W. Wisnicky.

Applications Pending

SECOND LISTING

(See June, 1932, JOURNAL)

Catalan, Nemesio, Los Banos, Laguna, P. I.
Dale, Chester N., Bureau of Animal Industry, Washington, D. C.
Etheridge, Joseph W., R. R. No. 2, Silver Spring, Md.
Kuhn, George A., D-4, Hillcrest Apts., South Saint Paul, Minn.
McCullough, J. Walter, Hanover, Pa.
MacDonald, Angus D., Metzerott Road, Berwyn, Md.
Mayfield, Orley J., Beltsville, Md.
Mohler, William M., 5508 Nebraska Ave. N. W., Washington, D. C.
Odom, Houston, Box 91, Auburn, Ala.
Port, Harry D., 310 Capitol Bldg., Cheyenne, Wyo.
Ruebush, Ephraim E., 3622 Georgia Ave. N. W., Washington, D. C.
Sherwood, James E., 6 Chestnut St., Suffern, N. Y.
Shook, Warren B., 1213 Gallatin St. N. W., Washington, D. C.
Smith, E. Barnwell, 222 C St. N. W., Washington, D. C.

The amount which should accompany an application filed this month is $7.50, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Wisconsin Veterinary Medical Association. Madison, Wis. July 5-6, 1932. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Minnesota State Veterinary Medical Society, combined with Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 7-8, 1932. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

North Dakota Veterinary Medical Association. Bismarck, N. Dak. July 11-12, 1932. Dr. Lee M. Roderick, Secretary, North Dakota Agricultural College, State College Station, Fargo, N. Dak.

Maine Veterinary Medical Association. State House, Augusta, Me. July 13, 1932. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 13-14, 1932. Dr. J. R. Stifler, Secretary, Lebanon, Ky.

Western New York Veterinary Medical Association. Chestnut Ridge Park and Hamburg, N. Y. July 14, 1932. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

New Jersey Veterinary Medical Association. Hotel Chelsea, Atlantic City, N. J. July 14-15, 1932. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Northwest Veterinary Medical Association. Hotel Vancouver, Vancouver, B. C. July 18-20, 1932. Dr. Clifford Ackley, Secretary, Winlock, Wash.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. July 20, 1932. Dr. E. E. Jones, 1451 Mirasol St., Los Angeles, Calif.

Missouri Veterinary Medical Association. Springfield, Mo. July 20-21, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Montana Veterinary Medical Association. Helena, Mont. July 21-22, 1932. Dr. Hadleigh Marsh, Secretary, Agr. Exp. Sta., Bozeman, Mont.

Connecticut Veterinary Medical Association. Bridgeport, Conn. August 3, 1932. Dr. Edwin Laitinen, Secretary, 993 Main St., West Hartford, Conn.

American Veterinary Medical Association. Atlanta Biltmore Hotel, Atlanta, Ga. August 23-26, 1932. Dr. H. Preston Hoskins, Secretary, 1230 W. Washington Blvd., Chicago, Ill.

ANTHRAX*

By N. F. WILLIAMS, *Fort Worth, Texas*

Chief Veterinarian, Live Stock Sanitary Commission of Texas

The control and eradication of anthrax in live stock is urgent from both economic and public health standpoints. The deplorable lack of consistent control is an evidence of man's age-old disregard of his own welfare. One of the oldest diseases known, anthrax was numbered among the plagues of Egypt, and has been recorded in almost every age since the history of the human race began. The disease was probably spread by venturesome humans who, overcoming the natural physical barriers of their time, carried the infection in their live stock and effects to the new lands that they discovered. Advancing from west to east, it progressively ravished the old world, its widespread dissemination over Europe being noted in the ninth and tenth centuries.

Vaughn suggests the probability that the extinction of certain species of animals may have resulted from this infection, and that were it not for some powerful antagonists, such as the *Bacillus pyocyaneus*, the pneumonia bacillus, and certain staphylococci and streptococci, the world might have long since been depopulated by the unopposed activity of the anthrax bacillus, the weapons of which are effective against so many species.

Competent authorities tell us that anthrax is considered the most typically infectious of all diseases, and is the first disease of man and animals shown to be caused by a microörganism. It is rod-shaped, generally square-ended, multiplies by transverse fission, and forms chains of bamboo pattern. It is probably the only aerobic spore-bearing bacillus of particular medical importtance, its spore being one of the most resistant forms of pathogenic bacteria.

When times are hard and circumstances unfavorable, the organism saves its life by passing into a resting stage, a spore form, in which condition it is only potentially alive and consequently requires neither air nor food. When times get good again, the organism reassumes the vegetative form, a fragment of the spore occasionally persisting on the end of the rod. If everything is favorable, it proceeds to multiply luxuriantly. The fact that

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

this vegetative form cannot live to advantage without oxygen is of great importance in the matter of control.

It is generally conceded that this bacillus was discovered by Bollender in 1849, and that in 1869 Davaine recognized its nature. Of Davaine's experiments, those in which he demonstrated that the blood of the anthrax-sick animal is not capable of transmitting the disease to others unless it contains the bacillus, are probably most important, because attention is specifically directed to the fact that the organism can be found in the blood of the animal at the hour of death and immediately thereafter, but not before. Because of its vital importance to orderly diagnosis, it may be pardonable to describe briefly the experiment which led to the demonstration of this fact.

Rabbit A was inoculated with anthrax blood. Forty-six hours later, examination showed no bacilli in the blood of rabbit A. At that time, 12 or 13 drops of blood were taken from the ear of this animal and injected into rabbit B. Nine hours later (55 hours after inoculation), the blood of rabbit A was reëxamined and found to contain a large number of bacilli. This blood was injected subcutaneously into rabbit C. One hour later (56 hours following inoculation), rabbit A died, and 24 hours later rabbit C died, while rabbit B remained free from infection. The fact here established is of great importance as a guide in the procuring of blood specimens for diagnostic purposes.

Koch Makes Discoveries

In 1876, the uniquely endowed Dr. Robert Koch made further discoveries in the anthrax field and somewhat cleared the situation by cultivating the organism from the blood and producing anthrax in susceptible animals by inoculation of those cultures. He worked out the life history of the organism, and formulated the requirements necessary to prove its specificity and to establish its definite relationship to the disease.

In 1877, another genius, Louis Pasteur, the chemist, discovered a means of protective vaccination by the use of attenuated anthrax organisms. The manner in which Pasteur demonstrated his discovery to an incredulous, hostile public was most dramatic and convincing. Of 50 sheep, 25 were vaccinated, and 25 were not. Two weeks later, the 50 were given a lethal dose of fully virulent anthrax, with the result that all of the unvaccinated sheep succumbed to anthrax, while the entire 25 vaccinated sheep remained well. Practically all anthrax vaccines used since Pas-

teur's unparalleled demonstration have been composed of more or less attenuated living organisms in spore or vegetative form. The quest is for an ideal immunizing agent, one from which the living organism has been excluded.

Joseph McFarland mentions bacilli presenting the morphologic and cultural characteristics of the anthrax bacillus, but devoid of any disease-producing power. Of these, *B. anthracoides*, *B. anthracis similis*, and *B. pseudo-anthracis* have been given special names. What relation they bear to the anthrax bacillus is uncertain. He concludes that these may be entirely different organisms, or they may be individuals whose virulence has been lost through unfavorable environment. This is important because of the all too prevalent belief that an accurate diagnosis of anthrax can be reached by microscopic examination of a blood-smear alone, and without animal inoculation.

As evidence that we are dealing with a most resistant pathogenic organism, against which the best methods of vaccination are only partially successful, authorities tell us that bouillon cultures of the vegetative form are sterilized by heat at 80° C. (176° F.), boiling for thirty minutes being necessary to insure destruction of the spores. Dry heat at 100° C. (212° F.) must be continued for two hours in order to destroy the vegetative form, while three hours at 140° C. (284° F.) are necessary to kill the spores.

Resistant to Cold

Resistant to heat, the organism is even more resistant to extremes of cold, as is shown so clearly by Dr. J. I. Quin, whom I am pleased to quote. In his thesis accepted for the degree Doctor of Veterinary Science, by the University of South Africa, Dr. Quin recited his efforts to attenuate virulent anthrax cultures by repeated freezing and thawing. A saline emulsion of a 12-hour agar growth of virulent anthrax organisms was placed in an ingenious apparatus consisting of two galvanized iron tubes, each closed at one end, the one suspended within the other. A small inlet in the outer tube was connected with a cylinder containing carbon dioxid under high pressure. Opening the cylinder caused the rapid escape of CO_2 from between the two tubes. This resulted in almost immediate freezing of the emulsion. After remaining frozen for ten minutes, the inner tube was removed and quickly immersed in water at 90° F. When liquefaction was complete, the freezing was repeated. Subcultures on nutrient

agar were made after every freezing and incubated at 37° C. (98.6° F.). This freezing and thawing was continued 24 times in succession.

After incubating the agar cultures for 24 hours, approximately equal amounts of growth (one platinum loop full) were removed and suspended in definite amounts of saline. This was subsequently injected into guinea pigs and rabbits. One cc of the 24-hour-old virulent agar culture killed the rabbit with anthrax in seven days, one guinea pig in two days, and the other in five days. One cc of material frozen 21 times killed the rabbit and the two guinea pigs in three days. As the "frozen 21" showed no signs of attenuation, the "frozen 23" culture was taken and frozen for two days and termed "frozen 24." Saline emulsions were made as above, and guinea pigs and rabbits injected, only with much smaller amounts. The rabbit injected with .001 cc of the emulsion died in three days, of anthrax. A guinea pig injected with .001 cc of emulsion was dead in two days, of anthrax. A guinea pig injected with .0001 cc of emulsion was dead in five days of anthrax. A guinea pig injected with .00001 cc of emulsion was dead in three days of anthrax. The experiment shows that the young 24-hour-old vegetative forms of virulent anthrax could withstand with impunity persistent rapid alternate freezing and thawing. Microscopic examination of smears made from the frozen material revealed not the slightest signs of disintegration, while the animal tests proved the absence of any attenuation having taken place.

Effect of Mercuric Chlorid on Spores

While Marshall states that a 1:1000 solution of mercuric chlorid will destroy spores in a few minutes, Geppert found that after from two to three hours in corrosive sublimate (1:1000), all spores are not killed. Investigation shows that when the temperature is under 12° C. (53.6° F.), there is no further development, and the sporulation ceases at temperatures below 14° C. (57.2° F.). Dr. Koch is said to have demonstrated that the anthrax organism in the properly buried carcass probably suffered the same fate as the other body cells. Frankel points out that the serious soil infection is the pollution of its surface by the bacilli-rich blood stools and urine of the live animal rather than the unopened or properly buried carcass. Were this otherwise, certain known anthrax areas would have become so thoroughly saturated as to defy immunization or to render the immunes and

the herbage of the pastures dangerous carriers of the organism to new fields and susceptible animals. Burial six feet deep is generally believed to preclude sporulation positively.

Although the study of anthrax has contributed most distinctly to our knowledge of bacteriology in general, a great many points concerning the *Bacillus anthracis* and the disease it produces remain unsolved.

We shall never know the thrilling story of the migration of this infection from the Valley of the Nile to the Mississippi Delta. Ships from the old world, clearing their cargoes through Gulf Coast ports, probably introduced anthrax to that part of our Coastal Plains that fringes the Gulf of Mexico, and is now known as natural anthrax country. Spanish troopers and adventurers active along the Rio Grande in the seventeenth century may have contributed infection in that direction. Just when or how the disease came to the southern states is not known. The statement that the United States Army posts in Texas suffered losses from charbon as early as 1868 is interesting, in view of Dr. W. H. Wray's conclusion that the disease was active in the Yazoo bottoms of western Mississippi in 1868, and from year to year up to 1890, when he, accompanied by Dr. John W. Connaway, of the University of Missouri, investigated such an outbreak in that locality.

Outbreak Near Fort Clark in 1880

Dr. Dickman, of the federal department, is said to have encountered a sharp outbreak of anthrax in the vicinity of Fort Clark, in 1880. Prior to 1868, and for some time after, there was no real market for Texas cattle. A few slaughtering plants were operated in the anthrax area, to which Coastal Plains cattle were driven and slaughtered for their hides and tallow, which products were shipped to market by boat. From 1871 to 1895, starting in the early spring, many cattle from the southern part of Texas were driven north to join the trail herds, aggregating 350,000 cattle, en route to northern markets each year. The fact that the cattle going north were moved from the polluted areas in February, well in advance of the anthrax season (July and August) no doubt saved the historic old trail and the states it traversed from serious infection, even though our boat-shipped hides may have contributed to the infection of New York and Pennsylvania meadows and pastures when tannery waste overflowed.

Little attention was given then to live stock losses on the Coastal Plains. The overabundance of cattle, the vastness of the operation on unfenced ranges which were worked but once or twice a year, precluded all thought of animal or range conservation. Carcasses of anthrax victims were left to nature's processes, the associated bones of mother and calf bleaching in the spring of another year, telling a story that cowmen eventually came to understand. The fencing of the ranges, with consequent congestion of the live stock population, changed all this and losses assumed economic importance as the cattle operation established itself on a firmer business basis.

In 1904, the Live Stock Sanitary Commission of Texas recorded an anthrax outbreak in southeast Texas near the Louisiana line. Again, in 1908, the disease was discovered in the lower marshy lands of the Gulf Coast. Stockmen were advised to vaccinate the cattle and burn the carcasses. The use of spore vaccines had been practiced in Texas for some time prior to the 1908 outbreak, and its use increased each year, although at times stockmen believed that outbreaks were caused that otherwise would not have occurred. In 1914, the disease occurred in 29 counties, and more extensive vaccination was recommended. New counties were added in 1915, State Veterinarian Christman succumbing to the disease during that outbreak. In 1916, 39 counties were ablaze, entailing a cattle loss of a half million dollars, and everybody's spore was in use in a hysterical effort to control the disease. Less use of vaccine and more attention to burning of carcasses was urged. Quarantines were declared, but enforcement suffered because of a lack of supporting funds necessary to maintain a field force.

Shortcomings of Spore Vaccines

Anthrax areas were gradually spreading. Spore vaccine, the sole immunizing agent available at that time, was in regular use, not by veterinarians, however, but by stockmen, cow-punchers and farmers, many of whom made their own diagnosis, all deaths in or near the Coastal Plains in July and August being charged to anthrax. When the vaccine failed, they applied their own remedies. Conflicting claims made for various spore vaccines directed the attention of the public to the shortcomings of the Pasteur product, and thus the daddy of all spore vaccines, the first to enter the field, was the first to be discredited. Because too much was expected of the spore vaccine in general, and to some extent no doubt because of careless handling, spore vaccine after spore

vaccine suffered a similar fate. A particular product used in a given locality one year would be emphatically repudiated the next. Year by year the spore vaccine was coping less satisfactorily with the anthrax situation, and the infected areas were spreading.

The year 1920 is outstanding in Texas history as one of widespread anthrax and the inauguration of an effort to control and eventually to eradicate it by means of close quarantine and the restricted use of immunizing agents, plus burning of carcasses. Anthrax is not ordinarily prevalent in Texas earlier than July, and rarely continues after August 15 (a period of 45 days in which flies are most active). This particular outbreak was discovered in a south Texas county on July 10, and almost immediately in nine adjoining counties not ordinarily active, while the area designated as heavily infected remained quiescent save for a sporadic case now and then.

Outbreak Traced to Spore Vaccine

Investigation of the 1920 outbreak revealed that in a certain county of more or less broken and inaccessible country it was a practice to vaccinate with anthrax spore vaccine when live stock losses occurred. From time to time live stock from that area were identified with sporadic anthrax in adjoining counties. Sometimes the sheep alone would suffer; again the loss would be limited to cattle, horses and mules. Again, all live stock species would be involved. The use of spore vaccine increased as the disease spread. This outbreak was so definitely traced to the use of spore vaccine that regulatory authorities recognized the necessity of immediate proscription of that product, the prompt burning of carcasses, and encouragement of the use of the sero-vaccine method of immunization, which was receiving the approval of bacteriologists and sanitarians at that time. The entire veterinary force of the Live Stock Sanitary Commission remained in the area of infection from July until September, when the outbreak subsided. Area quarantines were rigidly enforced, armed guards patrolled the highways, preventing the movement of live stock or contraband commodities. The further use of spore vaccine was prohibited and containers that were scattered on various premises were gathered and burned, as were remaining carcasses. With the use of the simultaneous method the veterinary practitioner came into the picture. Diagnoses were carefully made deaths from other causes differentiated, sanitary measures suggested and the confidence of the public was gradually restored.

Flies and buzzards in great numbers were active throughout this siege. That carrion-feeding animals, buzzards and carrion crows are carriers of anthrax infection has been conclusively demonstrated. In the anthrax areas of the South, the buzzard is a disseminator of the disease, and is a factor to reckon with during an outbreak. In our 1920 outbreak, 1600 buzzards were captured in one pasture in 48 hours by using a cleverly contrived net wire trap. As buzzards water at the same pools, ponds and tanks as do live stock, contamination of the water supply provides a ready means of infection transmission. The anthrax organism has been demonstrated in the droppings gathered and examined near buzzard roosts, suggesting the possibility of similar infection deposits at points between the carcass that furnished the feast and the roosting place of the birds.

Flies May Act as Carriers

The much beloved Dr. W. H. Dalrymple, who contributed so liberally to our knowledge of anthrax in the South, transmitted charbon to mice from the carcasses of rats by means of flies, demonstrating that flies are, in instances at least, carriers of anthrax infection. It is generally believed by veterinarians who are regularly and closely associated with anthrax outbreaks that the vicious, lancing horse- and deer-flies that literally swarm upon the disease-stunned anthrax victim as death approaches, are capable, at the time they abandon the carcass, of direct and immediate transmission of infection to the first animal they succeed in bleeding and, for a period at least, are carriers of the disease. The anthrax season in Texas coincides with that of fly activity, and an outbreak is shortened when intermittent heavy showers drown the larva and wash away the fly eggs from creek beds where they have been deposited. Dr. R. A. Caldwell, of California, calls attention to the age-old practice among stockmen of moving the herd or flock when sudden death losses occur. If the disease appears in fly time, the herd is moved at night, so that menacing, infection-charged flies are left behind. The blowfly is likewise a menace because of its direct contact with diseased tissue at a time when the blood is heavily charged with the microörganisms.

Entomologists, after placing suitable traps at various distances from the center of infection, exposed flies to aniline dye, immediately releasing them in an effort to determine the distance that infection might be carried by such means. Twenty-four hours

later these stained flies were found in all of the traps, some at a distance of fifteen miles, the surprising feature being that the extreme distance of flight was not with the wind alone, but in every direction.

Practitioners engaged in this outbreak conclusively demonstrated the curative value of anti-anthrax serum, by which means some valuable breeding stock was saved, as well as the lives of several persons who had become variously infected. Remarkable results followed the subcutaneous injections of 100 cc of serum while the infected animal's temperature was rising, and before it had reached its peak. With declining temperatures, results were not so good. Intravenous injection of greater amounts (250 to 500 cc) of serum, sometimes repeated, did revive cases that appeared quite hopeless. These latter subjects of happy termination were probably animals of high native resistance, capable of immediately utilizing to advantage the reënforcing antibodies provided in the serum.

Horses, mules, cattle and sheep were involved, and two stubborn points of infection were encountered in the Dry Frio Canyon that will figure prominently in a subsequent outbreak.

The Decline of Spore Vaccine

The year 1920 marked the decline of spore vaccine, which had served its purpose in a pioneer way, as it did the almost immediate popularization of the newer simultaneous method of immunization that has since served a useful purpose.

Five counties in the permanently infected anthrax area were involved in 1923. The disease did not occur in the 1920 area. Hay meadows, from which prairie hay is furnished on army contracts, are located in these counties. In November, 1923, anthrax appeared in a very acute form at Fort Bliss, in the Eighth Corps Area, 700 miles or more from this anthrax country, and was traced, to the satisfaction of Army officials, to old forage from affirmatively and permanently infected areas. In an effort to protect the Army Post, and indirectly the local civilian population, from such infection, it was proposed to exclude all hay and fodder that was harvested in anthrax areas. The hay contractors bitterly opposed such change of specifications, and were granted a hearing at Fort Sam Houston, February 11, 1924, at which much interesting evidence was presented. The possibility of infection by transmission on hay or forage was generally admitted,

although one hay contractor placed particular stress upon a statement of a colleague:

> The only way contagion could be spread would be by the rake picking up some dirt on the roots of the hay. I do not believe the contagion could be conveyed by the spores growing on the dry hay itself.

By carefully planned, practical experiments conducted between the years 1924 and 1926, the Department of Animal Pathology, Louisiana Experiment Station, demonstrated positively that anthrax spores could be carried from inoculated soil, on, but not within, forage plants. Of the plants used in this work and grown on inoculated soil, 92 per cent of the corn carried anthrax spores on all parts of the plant from the stems to the tips of the leaves, 80 per cent of the plants showed anthrax present on the first inch and one-half of the stems, while the tips of the leaves showed anthrax present on about 60 per cent. The number of anthrax colonies developing in the cultures varied from 1 to 15, with the greatest number on the lower parts of the plant. Eighty-four per cent of the oats showed the presence of anthrax spores distributed practically as on the corn, except that the percentage of the spores at the top of the plant was practically the same as that found near the ground (75 per cent). Practically the same number of colonies developed in the cultures as in the case of the corn.

Only 50 per cent of the rice plants showed the presence of anthrax spores, the percentage found on tops and bottom being practically the same. Few colonies developed in the culture. It was suggested that the decrease in infection compared to the other plants might be due to the flooding of the soil with water, following germination, to insure proper growth of the rice.

Of the bean plants, 100 per cent showed anthrax spores on all parts up to the second set of leaves, the cotyledons being covered with spores in every case. A large number of colonies developed in the cultures. The controls were negative. The work with Bermuda and bull grass was somewhat unsatisfactory because of the difficulty of inducing proper growth under laboratory conditions, but results were considered consistent with the other experiments, which indicated that the spores were carried on the surface of the plant and not in the plant tissue. Washing the plants with water for three successive days removed only a portion of the anthrax spores.

It was shown that anthrax had been transmitted to live stock in hay, in rice polish and in wheat straw from anthrax areas. The Louisiana experiment furnishes rather convincing evidence on this point, and is of direct interest to agricultural economists.

The hay contractors opposed the contemplated embargo upon the ground that proven cases of anthrax transmission by means of hay and fodder were too infrequent to justify embargoes against an industry of such magnitude as the growing, harvesting and marketing of hay and feed. Col. Ray J. Stanclift quoted the following statement of Dr. James Law, when stressing the necessity of preventing the pollution of pastures by tannery wastes, and advocating effective disinfection of hides regardless of the expense involved:

> If the trade cannot stand the expense, it has no right to exist where it is threatening, as it does, ruin, local and ultimately general, of agriculture, on which all other industries are based.

That statement holds good against hides, hay or any agency that is capable of producing or transmitting anthrax, including the spore vaccines. The only hope of eradicating anthrax, and we have a reasonable hope of doing that, lies in our general concurrence in Dr. Law's unimpeachable opinion on the matter of infection control.

Stock-Owners Become Discouraged

No outbreak of importance occurred until May 29, 1926, when several animals died in the Dry Frio Canyon from what was presumed to be anthrax. As had been the custom since the disastrous 1920 experience with spore vaccine, the animals in the neighborhood (the hot spot of 1920) were promptly vaccinated by the simultaneous method. Losses continued, the disease spreading to all parts of the canyon. There being no veterinarian in this locality, the outbreak was not reported to the Live Stock Sanitary Commission until June 6, immediately following which report, specimens favorable to a positive diagnosis of anthrax were procured. The failure of the simultaneous vaccination to hold in this instance so discouraged many of the stock-owners that they became dilatory about disposing of carcasses. In one instance a valuable horse, that died of anthrax on the morning of the seventeenth day following vaccination, was not disposed of before a wall of water sweeping down the canyon from a cloudburst in the hills carried it on, depositing the carcass 100 miles down stream, temporarily inundating the country along its course. As such high waters recede rapidly, live stock were soon grazing the overflowed pasture land again. Two weeks from the time the horse died and its carcass rode the cloudburst out of the canyon, anthrax in virulent form evidenced itself in the pasture where

the carcass had lodged, causing a serious cattle loss. All along the line of the high water sporadic anthrax has since occurred.

Area Quarantined

A quarantine prohibiting the movement of live stock within, from, or into the infected area was enforced, and stock-owners were encouraged to continue the use of the serum simultaneous method, or the anthrax aggressin which had then fought its way to the market after severe tests in the hottest anthrax field in Texas. When losses had not ceased at the end of the fourth week, animals already vaccinated with the simultaneous method were revaccinated with anthrax aggressin or No. 3 spore vaccine. One week after this was accomplished, losses ceased in the Dry Frio Canyon. Once the situation was explained to the stockmen in this locality, coöperation was of a high order. Herds heretofore not vaccinated were now treated at practically the same time; herds in which losses persisted were given individual attention. Further vaccination breaks from the seventeenth to the twenty-first day, where serum spore was used, were obviated by repeating the serum dosage on the fourteenth day. Anti-anthrax serum was administered to sick animals with favorable results. Unfortunately the disease had spread from the canyon early in the outbreak and now involved one-half of the county, menacing 10,000 horses, mules, cattle, sheep and swine. Deer, rabbits and squirrels died of anthrax and were not always discovered promptly enough to permit of advantageous disposal.

The people in this locality were reluctant to vaccinate, many of them resisting to the end, although they did burn carcasses. Others waited until the disease had attacked their herds with a consequence of high mortality. It was estimated that 2500 animals died from anthrax in this one county in an outbreak that started earlier and was more persistent than any that had previously occurred.

Fourteen Counties Involved

Although a block of 14 counties was involved in this instance, we have chosen to deal with a particular county as a unit, and to present the record of infected herds that were variously treated in this zone of primary infection because of its similar involvement in 1920, when the spore vaccine declined and the serum simultaneous method was popularized. The latter method of immunization almost completely superseded all other methods in

this area during the period from 1920 to 1926. Prompt burning of carcasses, regardless of the cause of death, was also religiously observed during that same period in this region, as it is today.

A report of 52 infected herds, involving 2516 animals, shows that 17 herds of 193 cattle that were not vaccinated suffered a loss of 28.5 per cent. Twenty-one infected herds of 1043 cattle, vaccinated with the simultaneous method and in many cases followed by No. 3 spore vaccine sustained a loss of 13.43 per cent. Fourteen infected herds, comprising 1280 cattle, treated with anthrax aggressin, suffered a 3.9 per cent loss. The report states:

> All herds were badly infected with anthrax and all in all everything was equal with the exception of the biologic used.

Anthrax was again prevalent in this county in 1927, but was held to a relatively small area by means of quarantine, the prompt burning of carcasses, and general use of anthrax aggressin. Aggressin has been used more or less generally as a prophylactic measure each year since, in this particular territory, and anthrax has not been active. The County Commissioners Court recently adopted a resolution commending the Live Stock Sanitary Commission for eradicating anthrax, which had proven so disastrous heretofore. The great danger now is that the stockmen will neglect to protect their live stock in the belief that the menace no longer exists, as has been done recently with blackleg, which is active again on premises where it has not appeared since vaccination with germless products became a routine some years ago. Hard times and low live stock prices, and the fact that the disease had not been prevalent for years tempted some stockmen to take a chance that has proven disastrous.

Anthrax Appears on Goat Ranch

At another point in the area of the 1926 infection, the disease appeared on a large goat ranch where a heavy loss was sustained within a few days. The ranch was quarantined, a burning crew was organized, the local veterinarian, aided by a member of the state veterinary force, vaccinated 5,000 goats with anthrax aggressin. Five head of goats died in the next five days, when losses ceased. The disease did not spread from this pasture, nor has it reappeared. Anthrax aggressin has been highly successful in protecting sheep even after the disease has appeared, when a further loss might be expected. Sheep upon a small ranch of mixed live stock were grazing a pasture in which two horses died suddenly from anthrax. The sheep were treated with anthrax aggressin and removed to another pasture, no loss being sus-

tained. The following year anthrax appeared among the sheep in a virulent form. Investigation disclosed that a shallow lake in the pasture where the horses had died the previous year had been reduced through lack of seasonal moisture, the sheep grazing on weeds that followed the receding water. The stockman took advantage of the occasion to remove skeletons that had been exposed and to clean out the water-hole. The sheep were vaccinated with anthrax aggressin, but were not moved from the pasture. The water-hole was fenced off and the pasture has been in constant use since, with no live stock losses from anthrax reported.

Revaccination Required

An outbreak of anthrax occurred this year which has been troublesome because of financial stress and a desire to avoid expense at almost any hazard. This outbreak was rather widespread before it was discovered by regulatory officials, and involved many farms but relatively few live stock. Spore vaccine was in use because it was the cheapest that could be procured, and the owners had been advised not to report losses for fear of being quarantined. The large ranchmen had vaccinated early with the simultaneous method or the anthrax aggressin, and did not suffer at this time, although the infection spread among the little operators throughout two counties. When discovered, the infection was so sharp as to necessitate rather extensive revaccination. In some cases a 10-cc dose of aggressin failed to stop losses entirely earlier than the nineteenth day. In one small herd of ten animals remaining after two had died from anthrax, and which the owner had vaccinated with 10-cc doses of anthrax aggressin, six died within ten days.

In a neighboring herd in which five had died and 31 were later vaccinated with aggressin, one animal died the day following vaccination, while one found sick recovered, following the prompt administration of 100 cc of anti-anthrax serum and an additional 50 cc on the next day. Two days later, another dead animal was found and burned. No further losses occurred in this herd. We are convinced that the regular 10-cc dose of aggressin will not give full protection when the organism is extremely virulent, as happens in some outbreaks, and doses of from 15 to 20 cc should be used. Losses occurred from the eighteenth to the twentieth day after vaccination with the simultaneous method, in some instances. If taken promptly, these cases responded satisfactorily to the serum treatment. A few herds that were treated with the

simultaneous method, and were given serum on the fourteenth day, did not suffer losses on the eighteenth to the twentieth day. In fairness to the products used after the spore vaccine was prohibited, it should be stated that with very few exceptions the herds treated had suffered more than one loss and on separate days, before vaccination was resorted to.

On one premise a horse had died and the carcass was fed to the hogs. Three hogs and the owner's dog died, the sudden death and swollen throat being suspicious of anthrax. A cow evidencing a swelling on the shoulder was found, and the case was diagnosed by a veterinarian as clinical anthrax. This cow recovered after having received 100 cc of anti-anthrax serum at once, and 50 cc on each of the two days following. Three horses and four cows were vaccinated with 15 cc of aggressin without further loss. Several instances in which the immunizing agent was abused were discovered. At the beginning of the outbreak a county agent administered 100 cc of liquid anthrax spore vaccine for southern use to a jennet which should have received 2 cc. The jennet died promptly and the ultimate loss on this farm was 10 out of 30 animals despite vaccination, or because of it.

A Serious Blunder

On another premise, where the simultaneous method was in use, the owner had injected the 1 cc of No. 2 spore vaccine when the cow broke out of the chute. She was returned and, after receiving the 10-cc injection on the other side of the neck, was released and driven to pasture with the other cattle. In preparing to vaccinate the horses, it was discovered that 10 cc of spore vaccine had been given to the obstreperous cow instead of 10 cc of serum as was intended. The owner could not remember which cow he had over-dosed until she was found dead the next day, having failed to withstand 11 cc of No. 2 spore vaccine. Three herds that had been vaccinated with anthrax aggressin 30 days previously were reported as suffering losses. It was discovered that the owners, who were all of one family, had for reasons of false economy used a dosage of only 5 cc, when the herds were vaccinated. Revaccination with 15-cc dosage stopped further loss. Eight animals in these herds evidenced anaphylaxis upon this occasion, but all recovered. All of which adds to the reasons why the practitioner should be actively associated with the vaccination of live stock, especially where a serious infectious disease is involved.

The first essential to the control of infectious live stock diseases is the destruction of the disease-producing germs or virus. Dr. James Law's admonition as to anthrax spore vaccine is here quoted:

> It can never be ignored that we are dealing with the living seed of a most deadly infection. Though robbed of a large part of its virulence by artificial culture at 42° C. (107.5° F.), yet many accidental conditions contribute to a relapse to its original potency, and when it has once killed a specially susceptible victim, the renewed virulence is usually persistent. The germs reinforced in potency in any such way are liable to be the starting points for dangerous infections in animals and permanent contamination of soils and water. With a wide application of the Pasteurean inoculation, the opportunities are not wanting, and with the free sale and distribution of the enfeebled germ, the evil may grow indefinitely. The method departs from the ideal one, which aims at a final extinction of the disease, and accepts in place a mere temporary protection of the generation which makes up the herd or flock at a given time, with no consideration for the generations that are to come after. Eradication of anthrax cannot always be secured, yet every effort should be made to attain it, and above all to check its infection on new land.

The epidemiology of anthrax was worked out sufficiently years ago to guide sanitarians in the control and eradication of this disease. Davaine demonstrated exactly when the organism could be found in the blood of the infected animal, thereby facilitating diagnosis. Dr. Koch established the procedure necessary to prove the specificity of the disease, a further and unimpeachable aid to diagnosis. He also demonstrated that the organism in the unopened carcass and six feet under ground suffered the same fate as the body cells, and did not form spores or pollute the soil. To incineration another dependable method of cadaver disposal thus was added. Louis Pasteur provided a means of temporarily immunizing a large percentage of animals vaccinated with attenuated germs at the expense of the more susceptible individuals and of premise infection. Despite its dangerous character, sanitarians for years tolerated the spore vaccine as a crutch for a crippled situation to lean upon and hesitated to kick the crutch from under, in the absence of a suitable substitute. That contingency no longer exists. We do not believe that the objections raised against spore vaccine in the past have been in any manner mitigated. As long as a spore vaccine of sufficient virulence to be effective is in use, anthrax can never be eradicated.

Texas experience has convinced us that as far as dependable immunization is concerned no form of anthrax vaccine, serum-spore included, is at all superior to the proper use of aggressin; or where indicated, of aggressin with serum. We believe that the 10-cc dosage should be increased to 15 cc, and that vaccination should be repeated in extremely virulent outbreaks. We do not

believe that there is at present any method of anthrax vaccination whereby a single injection will produce a lasting immunity against serious infection. We do believe that in years of mild infection a suitable dosage of aggressin is fully as effective as any form of serum-spore vaccine. The aggressin approaches the ideal, germless, immunizing agent that has been looked for so long, and within a relatively short time will increase its efficiency or in turn give way to a better germless method of protection.

The control and ultimate eradication of anthrax depends, first, upon correct diagnosis, supported by positive cultural, inoculation, and microscopic proof; second, upon prompt incineration or burial of carcasses; third, upon the immediate treatment of sick animals with anti-anthrax serum and the protection of susceptible exposed animals by means of a germless immunizing agent; fourth, upon rigid enforcement of a quarantine that will positively prohibit the movement of live stock or contraband commodities from, within, or into the infected area.

In his presidential address to the U. S. Live Stock Sanitary Association, in Chicago, in December, 1928, Dr. C. A. Cary, whose method of anthrax control in Alabama approaches eradication, said:

> We wanted to stamp out tick fever by the stamping-out method, and we are doing that in great areas, and it is not returning unless we return some of the factors that carry the infection. I don't know of any disease that I have worked with in which we have accomplished so accurately and completely the process of eradication. Of course, this means the control and prevention of the disease, but in the end it is eradication. I want you to get that point.

Dr. Cary has sounded the keynote to the final solution of the vexing anthrax problem. Eradication can never be accomplished as long as a living spore remains, be that spore ever so attenuated, impoverished and degraded. We must confess that we have not heretofore done justice to the eradication feature so essential to the future welfare of agriculture and human happiness, and should rededicate ourselves to the completion of a task with which we have unfortunately temporized too long.

Cedar Rapids to Protect Milk Supply

The City Council of Cedar Rapids, Iowa, is considering the advisability of making changes in the ordinance regulating the sale of milk locally, with a view to protecting the public from any possibility of danger from undulant fever transmitted through the medium of raw milk.

SALMON POISONING: TRANSMISSION AND IMMUNIZATION EXPERIMENTS*

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Introduction

Pernot[1] seems to have conducted the first recorded experiments concerning the transmission of salmon poisoning in dogs. He claimed the disease was caused by an amoeba, that it could be transmitted through blood injection, and that it could be cured through the administration of calomel.

Ever since the first studies of this disease by members of the staff of the Department of Veterinary Medicine of the Oregon Agricultural Experiment Station showed that it was associated with infestation with the intestinal fluke *Nanophyetus salmincola*, Chapin, it has been realized that this malady has many characteristics of an acute infection. The definite incubation period, the sudden onset, the severe systemic reaction, the rapid course, and the definite immunity in those dogs which recover, are all suggestive of an infectious disease. Among the first experiments were attempts to transmit it by some other method than feeding fish and to establish immunity through some other means than recovery from an attack of the disease. Donham, Simms and Miller[2] failed to produce salmon poisoning through feeding a susceptible dog both mature flukes and the intestinal content from a dog dead of the trouble. Their preliminary bacteriological studies gave negative results. They failed to produce immunity through injecting blood from an immune dog. Donham[3] failed to transmit the disease through a single intraperitoneal injection of 8 cc of blood from a sick dog into a susceptible one. He did not isolate any pathogenic organisms from any of the six dogs which were studied bacteriologically. He produced symptoms comparable to those of salmon poisoning in three of four susceptible dogs injected intraperitoneally with ground-up mature flukes, but failed to produce such symptoms in one immune dog so injected. Simms, Donham, Shaw and McCapes[4] injected fresh untreated flukes intraperitoneally in 19 susceptible dogs,

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17 of which developed symptoms, with 14 of them dying. The three which recovered were apparently immune. They failed to produce any reactions through the intraperitoneal injection of either previously heated or previously frozen flukes. One of the two dogs which they injected intraperitoneally with blood from a sick dog developed symptoms similar to salmon poisoning and died. In their experiments, kennel exposures uniformly failed to transmit the disease. They did not produce an immunity against the trouble through repeated administration of sublethal numbers of encysted flukes.

Suckley,[5] in 1855, reported that a single attack resulted in an immunity, but stated that "scarcely one in ten recovers." Donham, Simms, and Miller[2] confirmed this observation concerning the high mortality. It was evident from their studies that it was not practicable to immunize against the disease through exposing animals to it and expecting them to recover.

This paper records further attempts at transmitting salmon poisoning and immunizing against it.

Transmission Studies

1. Kennel exposure: During the seven years the disease has been under observation it has been the usual practice to pen several dogs together. Probably more than half of the two hundred odd susceptible dogs which have been studied have been penned with dogs which were affected with salmon poisoning. In no instance has there been any evidence that the disease was spread by such contact. The following is a typical example of such exposures:

Dogs 294 and 295, litter mates, whelped May 17, 1931, were infected with the disease; dog 294 by feeding parasitized fish on July 17, 1931, and dog 295 by an injection of virulent blood on July 9, 1931. They were placed with five untreated litter mates in an out-of-doors pen about 20 feet square. All the dogs were fed and watered from the same utensils. Dog 294 developed a typical temperature reaction on the fifth day and died on the eighteenth day following the administration of parasitized fish. Dog 295 developed a temperature reaction on the seventh day and died on the thirteenth day after the blood injection. None of the five litter mates developed any symptoms.

2. Injection of blood of dogs sick with salmon poisoning: A disease apparently identical with the one which is produced through feeding parasitized fish has been consistently trans-

mitted through either intraperitoneal or subcutaneous injection of either whole or defibrinated blood from sick dogs. Blood from five fish-fed dogs has been tested with positive results in every instance. In three of these tests the disease was transmitted from the injected dogs to others through a second transfer of blood. In one of the two others a single attempt at transmission to a second dog failed. In the fifth case no second transfer was tried.

In one instance (table I), the disease was passed through 16 successive dogs before it was allowed to die out.

TABLE I—*Transmission experiments (causal agent—strain II).*

Dog	Intraperitoneal Injection (Date)	Blood	
		Amount (cc)	From Dog
239	12- 8-30	3.5	237*
240	12-16-30	3.5	239
243	12-23-30	2.0	240
244	12-29-30	2.0	243
246	1- 5-31	2.0	244
252	1-14-31	3.0	246
255	1-22-31	4.0	252
256	1-31-31	2.0	250
258	2- 9-31	2.0	256
249	2-18-31	2.5	258
259	2-25-31	4.0	249
242	3- 7-31	0.1	259
263	3-21-31	5.0	242
265	4- 2-31	0.1	263
266	4-11-31	2.0	265
270	4-21-31	0.1	266

*Fed fish, December 1, 1930.

All the dogs in this series developed symptoms typical of salmon poisoning. Four of the 16 were destroyed, one recovered, and 11 died. One-tenth of a cc of blood was the smallest amount injected. This was apparently as virulent as the larger amounts.

All told, the disease has been produced in 30 dogs through either intraperitoneal or subcutaneous injections of virulent blood. Seven of these were destroyed during the course of the malady, 20 died at from the eleventh to the nineteenth day, and 3 recovered. The symptoms were apparently as severe in those that recovered as in the ones that died. They showed a complete loss of appetite for approximately a week, a very marked loss of flesh, a severe diarrhea, and a muco-purulent conjunctivitis.

A single dog (285) was injected intraperitoneally with 2 cc of defibrinated blood which had been taken from a sick dog and stored for 36 days at about 37° F. No symptoms developed

following this injection. This dog later succumbed, after being injected with fresh blood from a sick dog.

Ten dogs immune to salmon poisoning, as determined by feeding tests, were injected with virulent blood. In no instance was there a reaction typical of salmon poisoning.

3. *Injection of organisms isolated from sick dogs:* Results of attempts at finding the causal agent through microscopic examnation have been negative. These have included both dark-field and transmitted-light studies of both blood-cells and blood -serum and studies of stained blood-films.

Likewise, attempts to recover microörganisms from affected dogs have usually given negative results. The media inoculated have included broth, serum broth, glycerin agar, liver-infusion agar, blood agar, serum agar and Löfflers blood-serum. Heart-blood, spleen, lymph-glands and kidneys have been used for inoculations. Both aerobic and anaerobic methods of culture have been attempted. Two organisms, one from the spleen of dog 239 and the other from the heart-blood of dog 240 were obtained. Both failed to produce symptoms of the disease or any protection against subsequent exposure when injected intraperitoneally.

The different special types of media have not been used in these studies.

4. *Injection of filtrates:* Only four attempts have been made to pass the virulent agent through filters. Results of injecting such filtered material into susceptible dogs have been uniformly negative. (See table II.)

TABLE II—*Injection of filtrates.*

DATE	DOG	MATERIAL	FILTER	INTRAPERITONEAL INJECTION (CC)	RESULT
12–16–30	242	Flukes ground in 85% NaCl solution	Mandler (medium porosity)	8	Negative
1– 5–31	247	Blood-serum of sick dog	Mandler (medium porosity)	5	Negative
2–25–31	260	Blood-serum of sick dog	Seitz	5	Negative
8– 5–31	307	Blood of sick dog diluted with 10 parts distilled water	Seitz	5	Negative

5. Feeding blood from a sick dog: Following the demonstration of the fact that the injection of blood from a sick dog would result in a disease comparable to salmon poisoning, a single susceptible dog (254) was fed 10 cc of virulent blood. The animal remained normal until it was later given an intraperitoneal injection to test its susceptibility. Following this it developed typical symptoms and died.

6. Injection of encysted cercariae from fish: Two dogs (236 and 268) were injected intraperitoneally with cercariae obtained from fish kidney. In one instance the parasites were obtained from a chinook salmon (*Onchorynchus tschawytscha*) and in the other from a cut-throat trout (*Salmo clarkii*). Both animals developed symptoms typical of salmon poisoning and one of them died. The one that recovered, a kennel-raised dog with no previous opportunity to eat fish, was later fed parasitized trout with no indications of disease following.

7. Injection of rediae and cercariae from snails: In two instances rediae and cercariae of the salmon-poisoning fluke were obtained by crushing parasitized snails. They were suspended in .85 per cent NaCl solution and injected intraperitoneally in dogs 236 and 305. No untoward effects were noted following these injections. One of these dogs (236) later reacted when injected with cercariae from fish. The other, a kennel-raised dog which had never been fed fish, has not been tested for susceptibility.

8. Injection of blood from a parasitized fish: A single young, kennel-raised dog (309) was injected intraperitoneally with 2 cc of blood from a chinook salmon. The fish had been dead less than 20 hours and was in good condition. It was heavily infested with viable encysted cercariae. No reaction was observed following this injection.

9. Injection of blood from a raccoon: Negative results were obtained in a young, kennel-raised dog (306) which was injected intraperitoneally with 0.5 cc of blood from a raccoon (*Lotor procyon pacifera*) which had been fed parasitized fish on two different occasions and had passed eggs of the salmon poisoning fluke following both feedings.

10. Injection of virulent blood into other species than the dog: One guinea pig, 1 white rat, 1 rabbit, and 1 raccoon were injected intraperitoneally with blood from a sick dog, the first three receiving 1 cc each and the raccoon 2 cc. Results were negative. This confirmed results of feeding tests previously reported by Simms, Donham, Shaw and McCapes,[4] in which they found

that the parasites developed to maturity, but did not cause symptoms in other carnivores than those of the dog family.

Immunization Studies

1. Injection of virulent blood: As reported above, three of the susceptible dogs injected with virulent blood recovered. They later both ate parasitized fish and received injections of virulent blood without developing any symptoms of salmon poisoning.

2. Injection of virulent blood and of blood from hyperimmune dogs: Two dogs weighing about 40 pounds each were used as sources of hyperimmune blood in attempts at producing immunity through injection of both virulent blood and hyperimmune blood. One of these (231) had recovered from an intraperitoneal injection of flukes and had later been fed parasitized trout without symptoms developing, and the other (251) was said to have recovered from an attack of salmon poisoning following the consumption of parasitized fish.

Hyperimmune blood was produced through injecting virulent blood into the peritoneal cavities of these immune dogs. The amount used at each injection varied from 60 cc, or 1.5 cc per pound body weight, to 200 cc, or 5 cc per pound body weight. In some instances, the blood was defibrinated before injection into the dogs being hyperimmunized, and in others it was injected whole, as soon as it was collected. The virulent blood was collected from sick dogs at from the seventh to the tenth day after feeding fish or injection with virulent blood. At this time the temperature was about at the peak. These dogs were bled either from the carotid or from the heart according to Lockhart's[6] technic. The hyperimmune dogs were bled from either the jugular or the recurrent tarsal vein.

Twenty-two dogs, 20 of which had known negative history in so far as salmon poisoning was concerned, have been injected with both virulent and hyperimmune blood. (See table III.)

The first four animals in this series were treated with hyperimmune blood collected from dog 251, following injections of not to exceed 100 cc of virulent blood, or not more than 2.5 cc per pound body weight. While three of these four dogs died following the two injections, dog 245 lived nine days longer than its control and dog 254 lived five days longer than its control. The control for dog 260 was destroyed in order that its virulent blood could be used for hyperimmunization.

TABLE III—*Immunization experiments.*

Date (1931)	Dog	Age	Virulent Blood		Hyper-immune Blood (I) (cc)	Source (Dog)	Interval after Hypering (Days)	Virulent Blood Injected at Last Hypering (cc)	Result of			
			Amt. (cc)	How					Injection of Virulent and Hyperimmune Blood	Injection of Virulent Blood in Control	Subsequent Feeding of Fish	Feeding Fish to Control
1–31	245	3 mos.	2.0	I	16		9	100	D. 21st day	D. 12th day		
3– 7	242	Mature	0.1	I	40		10	60	Lived	D. 13th day	Lived	D. 14th day
3– 7	254	4 mos.	2.0	I	40		10	60	D. 18th day	Note 1		
4– 2	260	4 mos.	1.0	I	40		26	60	D. 21st day	Destroyed		
4–21	271	2 mos.	0.1	I	23	251	10	200	Lived	D. 13th day	Lived	None
4–21	272	2 mos.	0.1	I	40		10	200	Lived	Note 1	D. 19th day	None
4–21	273	2 mos.	0.1	I	40		10	200	Lived	Note 1	Lived	None
5– 5	277	Mature	0.1	I	30		25	200	Lived	Recovered	Lived	None
5– 5	278	2.5 mos.	0.1	I	40		25	200	Lived	Note 1	Lived	None
5– 5	279	2.5 mos.	0.1	I	25		25	200	Lived	Note 1	D. 14th day	None
6–22	281	Mature	1.5	S	40		48		Lived	Recovered	Lived	Both died
6–22	286	Mature	1.5	S	40		48		Lived	Note 1	Lived	Note 2
6–22	287	1 yr.	1.5	S	40		48		Lived	Note 1	Lived	Note 2
6–22	288	1 yr.	1.5	S	40	231	48	200	Lived	Note 1	Lived	Note 2
6–22	289	1 yr.	1.5	S	40	and	48	each	Lived	Note 1	Lived	Note 2
6–22	290	3 mos.	1.0	S	25	251	48		Lived	Note 1	Lived	Note 2
6–22	291	3 mos.	1.0	S	25		48		Lived	Note 1	Lived	Note 2
6–22	292	3 mos.	1.0	S	25		48		Lived	Note 1	Lived	Note 2
7–17	299	2 mos.	2.0	S	20		25	200	Lived	D. 14th day	Lived	None
7–17	300	2 mos.	1.0	S	30	231	25	200	Lived	Note 1	Lived	None
7–27	302	2 mos.	2.0	S	30		35	200	Lived	D. 18th day	Lived	None
7–27	303	2 mos.	2.0	S	20		35	200	Lived	Note 1	Lived	None

I = intraperitoneally.
S = subcutaneously.
D. = died.

Note 1 = same control as above.
Note 2 = same controls as above.

There were no fatalities among the last 18 dogs so injected. The source of the hyperimmune blood used in these animals was either pooled blood from dogs 231 and 251, or blood from only one of these animals. In every case in this group the dog from which the hyperimmune blood was obtained had received an injection of at least 200 cc, or 5 cc per pound body weight, of virulent blood not less than 10 days nor more than 48 days preceding the bleeding.

The first ten dogs in the series were injected intraperitoneally with both virulent and hyperimmune blood, the time elapsing between the two injections never exceeding five minutes. In the last 12 animals, virulent blood was injected subcutaneously, and hyperimmune blood intraperitoneally.

The 19 dogs which survived the injections were fed parasitized fish. There were two fatalities among the seven which received both injections intraperitoneally and none among the twelve which received the virulent material subcutaneously and the hyperimmune blood intraperitoneally. It was determined that flukes developed to maturity in all these 19 animals through finding fluke eggs in their feces. Four of them were fed parasitized fish a second time. Virulent blood was injected intraperitoneally in four of these immunized dogs.

Seven of the 22 dogs in this series were mature animals and 15 varied in age from eight weeks to four months. The five fatalities were all among the younger dogs.

A systemic reaction was observed in nearly all of the dogs which received the injections of virulent and hyperimmune blood. This consisted in an elevation of temperature, loss of weight, and usually a partial loss of appetite. In most of the animals there was some diarrhea, with mucoid or muco-sanguineous feces. In some instances, the animals refused all food for one or two days. The animals usually remained bright and active even when the temperature was considerably above normal. These symptoms usually appeared 7 to 10 days following the injections. From 10 to 15 days were required for the animal to return to normal. The control dogs exhibited rises in temperature earlier than did the animals receiving hyperimmune blood with the virulent blood, and in most instances the temperatures of the controls rose to a higher point than was recorded for the simultaneously injected animals.

In all instances, at least 17 days elapsed between the injections and the feeding of fish. In those dogs which were still showing a

systemic reaction at the end of 17 days, feeding of fish was delayed until this reaction had disappeared. Eleven of the 19 simultaneously injected dogs which were later fed parasitized fish showed reactions very similar to those recorded following the injection of both virulent and hyperimmune blood. None of the four which were given a second feeding of fish showed any symptoms following this second ingestion of parasites. The injection of virulent blood in a group of four immunized animals did not result in any systemic reaction.

Discussion

While it has been shown that the malady is a transmissible one, one, both the nature and the origin of the causal agent remain unknown. Neither the bacteriological examinations nor the filtration experiments have been sufficient to allow definite conclusions to be drawn. Likewise, the examinations of both fresh blood and stained blood-films have not been repeated often enough to eliminate the possibility of the presence of a visible organism.

The origin of the infectious agent is still more puzzling than its nature. Four general groups of parasite-borne infections have been recognized and described; namely, those such as malaria, in which the vector must attack an infected individual and then a susceptible one of the same species in order to spread the disease; those such as sleeping sickness, in which the vector attacks an infected individual and then may act as a carrier to infect an individual of a different species; those such as Rocky Mountain spotted fever, in which the vector lives one part of its life as a parasite on an infected animal and then attacks a different species as a host for another part of its life, carrying the disease to the second host; and those like Texas fever, in which the mature female, which has fed on an infected host, passes the infection through the egg to the offspring, which transmit the disease to susceptible individuals of the same species as the original host of the mother.

The fluke, which has proved to be the carrier of salmon poisoning, cannot pass from one host to another as can the parasitic arthropods, such as malaria-bearing mosquitoes or sleeping-sickness-bearing tsetse flies. If it transfers the causal agent from one of its intermediate hosts, the condition is essentially different from the transfer of Rocky Mountain spotted fever. The tick, which carries this fever to the human, feeds on another mammal

to become infected, while the intermediate hosts of the salmon-poisoning fluke, the snail and the fish, are both cold-blooded animals. If the infection is transmitted through the egg, the procedure is much more complex than in the case of Texas fever. The flukes which transmit the disease to dogs have very probably originally come from raccoons, as these are the only carnivores which seem prevalent enough in the salmon-poisoning areas to infest the snails of the streams. It would be necessary for the causal agent to pass from the miracidium, which hatches from the egg, to the rediae and then to the cercariae as the fluke develops in the snail. It is very probable there are at least several generations of rediae, as a single snail has been found to contain above 80,000 cercariae.[7] It seems unlikely that such gross infestation would occur unless the invading miracidium could multiply through several generations of rediae.

The theory that the fluke receives the causal agent from some one of its hosts and transfers it to the dog must be recognized as a probability. Preliminary injections of rediae and cercariae obtained from snails, of fish blood, and of raccoon blood, all of which have given negative results, might indicate that none of these is concerned as a carrier of the disease in question. It is recognized, however, that these injections have not been of sufficient number to allow any conclusions to be drawn. The setting up of the disease in dogs which were injected intraperitoneally with encysted cercariae from fish has done no more than prove that this stage of the fluke carries the causal agent. This fact had already been established through feeding encysted cercariae.

Still another possibility is that the infection is primarily a non-lethal one of the fluke and that the canine family is susceptible as an accidental host.

A great deal more work needs to be done before there will be available sufficient knowledge concerning immunization. The age at which dogs can be immunized with greatest safety, the best methods of producing hyperimmune blood, the minimal amount of such blood required to protect a dog, the optimal amount of virulent blood to be injected with hyperimmune blood, methods of preserving both virulent and hyperimmune blood, the minimal amount of virulent blood which will produce the disease, and the length of time between the injection of virulent and hyperimmune blood and the development of immunity need to be determined.

Perhaps the feeding of either parasitized fish or encysted cercariae obtained from such fish may be substituted for the injection of virulent blood in the immunization process. If so, it will materially lessen the complexity of the method.

The discovery of a worm-borne infectious disease and the production of an immunity against it leads to many interesting suggestions.

It is within the realms of possibility that the causal agent, transmitted by a worm, is biologically different from any of the groups heretofore established as causing transmissible diseases. Whatever the nature or the origin of the disease-producing agent, it would seem strange if this particular fluke was the only one of the entire group of parasitic helminths which is capable of carrying an infectious agent. It may be reasonable to expect that other worm-borne infections will be discovered. Perhaps at least some of the ill effects which are associated with infestation with the various helminths may ultimately prove to be the direct result of worm-borne infections.

Summary

1. Salmon poisoning has been transmitted from sick to susceptible dogs through blood injections. It has apparently been produced through the injection of encysted cercariae of the salmon-poisoning fluke obtained from parasitized fish.

2. Neither the nature nor the origin of the causal agent has been discovered.

3. Dogs have been immunized against the disease through the simultaneous injection of virulent blood and hyperimmune blood.

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Maryland Meeting Postponed

According to advices received from Dr. E. M. Pickens, there will be no summer meeting of the Maryland State Veterinary Medical Association. Plans are being made to hold a meeting some time in November to take the place of the 1932 semi-annual meeting and the next regular annual meeting.

A COMPARISON OF THREE METHODS OF TESTING FOR PULLORUM DISEASE WITH FINER INTERPRETATIONS OF READINGS ON THE OLD TUBE AGGLUTINATION TEST*

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The various serological tests for pullorum disease have been the subject of prolonged research during the last six or seven years, and various methods of testing have been advocated from time to time. At the present time, there is considerable difference of opinion in regard to the relative merits of the tests for this disease in fowls. Most of the experiment stations are agreed that so far the old tube agglutination or long method has been found to be the most dependable for the detection and eradication of pullorum disease from farm flocks.

The pullorin or wattle test, which, a few years ago, enjoyed rather short popularity as a means of diagnosing pullorum disease, is at present not recommended, and most research workers have turned their attention to other methods of detection of the disease.

The rapid serum and whole-blood tests have received considerable attention by various workers in the United States and other countries. One experiment station, at least, is using the rapid serum test as a means of eradicating pullorum disease from farm flocks and the reports to date are favorable to this test as a means of eradicating the disease.

The rapid whole-blood test also has been studied by several workers. In 1929, Bunyea, Hall and Dorset[1] described a whole-blood test by which they claimed that the selection of healthy birds could be made from a diseased flock with one handling and with the elimination of doubtful reactors frequently encountered in the slow test. The authors of this article found the results to check very closely with those obtained with the widely-used tube agglutination test. They further state, however, that it is their hope that the various investigators in this field may be sufficiently interested to compare this simplified test with methods now in use, and that in this way only will it be possible to determine the real value of this new method.

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

Schaffer, McDonald, Hall and Bunyea[2] report on the use of a stained antigen for the rapid whole-blood agglutination test:

> During the past two years the writers have made comparative tests of the rapid whole-blood method, using stained antigen, and of the tube method. The results were checked in some cases by bacteriological examination at autopsy. Results of this work indicate that the new stained antigen is a reliable diagnostic agent. For more extensive tests, this antigen was also submitted to interested investigators in various parts of the country. The favorable comments received as result of these coöperative efforts, together with their own experience, have led the writers to publish this brief announcement in the hope that it may stimulate further critical examination of the diagnostic value of the new antigen.

The authors state that they do not advocate that the tube agglutination method be discarded until a large-scale use of the new method by a number of different men has firmly established the superiority of the whole-blood test. They say that the tube method, though it has its drawbacks, has given good results in controlling losses from the disease. They state also that the stained antigen for the rapid whole-blood agglutination test for pullorum disease deserves extended field trials.

Coburn and Stafseth[3] report on a rapid whole-blood field test for pullorum disease, using stained antigen modeled after the Huddleson-Abell method of preparing antigen for the rapid serum agglutination test for abortion. This is a preliminary report in which they state that their stained antigen, used with whole blood for the rapid agglutination test, is practical as a field test for pullorum disease. They say:

> With minor changes in the procedure as outlined by Huddleson and Abell, an antigen was prepared for use with whole blood which showed very close agreement with the tube method in dilutions higher than those commonly used in the slow test, when compared in repeated tests of a large number of samples from commercial and Michigan State Experiment Station flocks. This close agreement in the low dilutions and the high sensitiveness indicated that finer distinctions were possible with this method than with the slow test, which fact was borne out in the culture work conducted on postmortem.

In a personal communication of February 25, 1931, at which time Dr. M. Dorset shipped us the experimental stained antigen, together with directions for its use, he has the following to say in regard to this method of testing:

> I may say that we believe this rapid blood method, which can be carried out on the farm if desired and the birds culled at the time of the test, thus avoiding a second handling, will check very well with the tube method where serum is used in dilutions of 1:50 and 1:100, provided the operator does not attempt to read the stained antigen test too closely. Of course, the best technic and the most effective interpretations of the results will be obtained only after prolonged use. The tendency of the men here who have used this test extensively is to disregard reactions which are doubtful as well as those which require a long time (5 minutes) to become manifest. More work is being done here in postmortem examination of reacting birds, to check up in a measure on the real significance of the reaction.

From the discussions I have had with those who are better versed in this field than I, I have a very strong impression that owing to the cumbersomeness of the tube method, it is doubtful whether bacillary white diarrhea can successfully be cleaned up by that method. A rapid test such as the one I am asking you to try would, if successful, enormously facilitate the testing of flocks.

This paper is presented to show the comparative results of three methods of blood testing; namely, the old tube test, the rapid serum test, and the rapid whole-blood test, using a stained antigen furnished by Dr. Dorset. The results which follow show, to our mind, what we might expect from the use of these three tests in the eradication of pullorum disease from farm flocks. In other words, this is a practical comparative test of the three methods to determine which one in our hands would yield the best results. The author realizes, in this investigation, that the observations on these three tests are limited and that perhaps on a series of flocks the results might vary somewhat from those obtained in this particular flock.

A flock of 259 birds was selected for making this test. It was one on which a previous test with the tube method had shown considerable infection, but none of the reactors were removed. Every effort was made in all cases to carry out each test carefully and accurately, according to directions.

Antigens Used in Test

The antigen used on the rapid whole-blood test was secured from Washington as stated. The rapid serum test antigen was prepared by this laboratory, according to standard methods. The antigen for the tube test was our regular antigen which is being used for the detection and eradication of pullorum disease in Missouri.

In interpreting the results of the test, the instructions in regard to the whole-blood test were carefully followed. The results of the tests on the 259 birds were as follows: Forty-seven birds gave a positive reaction to all three methods, 94 were positive to the tube test, 75 to the rapid serum test, and 63 to the whole-blood test. Twenty-three birds were positive to the tube test and not to other methods, 8 birds were positive to the rapid serum test and not to the others, and 6 birds were positive to the whole-blood test and not to other methods. In all, there were 111 birds positive to one or more tests.

Autopsy of Birds and Cultural Results

The positive birds were purchased from the owner for bacteriological examination. Each of the birds was examined

TABLE I—*Showing the reactions of the 111 birds that reacted to one or more of the tests, with the gross lesions and bacteriological findings.*

BIRD	TUBE	RAPID SERUM	WHOLE-BLOOD	GROSS	CULTURE
A2211	+	+	+	Ov +++	+
A2217	+	+	+	Ov ++++ Y	+
A2220	+	+	+	Ov +	+
A2226	+	+	+	Ov +	+
A2228	+	+	+	Ov ++++	+
A2305	+	+	+	Ov ++	+
A2315	+	+	+	Ov ++++	+
A2333	+	+	+	Ov +++ Y	+
A2359	+	+	+	Ov ++++	+
A2366	+	+	+	Ov ++++	+
A2374	+	+	+	Ov ++++	+
A2379	+	+	+	Ov +++	+
A2385	+	+	+	Ov ++++	+
A2393	+	+	+	Ov ++++	+
A2414	+	+	+	Ov ++	+
A2416	+	+	+	Ov ++++	+
A2423	+	+	+	Ov ++	+
A2440	+	+	+	Y	+
A2472	+	+	+	Ov ++	+
A2479	+	+	+	Ov +++	+
A2485	+	+	+	Ov ++ Y	+
A2500	+	+	+	Ov ++	+
A2507	+	+	+	Ov +++	+
A2509	+	+	+	Ov ++ Y	+
A2515	+	+	+	Ov +++ H	+
A2516	+	+	+	Ov ++	+
A2522	+	+	+	Ov ++++ Y	+
A2525	+	+	+	Ov ++++	+
A2534	+	+	+	Ov ++ Y	+
A2544	+	+	+	Ov ++	+
A2561	+	+	+	Ov + Y	+
A2564	+	+	+	Ov ++++	+
A2577	+	+	+	Ov +++	+
A2583	+	+	+	Ov +++	+
A2595	+	+	+	Ov ++	+
A2600	+	+	+	Ov + Y	+
A2604	+	+	+	Ov +++	+
A2610	+	+	+	Ov ++	+
A2518	+	+	+	Normal	+
A2336	+	+	+	Ov +	—
A2489	+	+	+	Ov +++	—
A2490	+	+	+	Ov +	—
A2523	+	+	+	Ov ++++	—
A2570	+	+	+	Ov +++	—
A2450	+	+	+	Normal	—
A2502	+	+	+	Normal	—
A2434	+	+	+	Normal	—
A2317	+	+	—	Ov ++++	+
A2343	+	+	—	Ov ++++	+
A2353	+	+	—	Ov +	+
A2425	+	+	—	Ov +	+
A2429	+	+	—	Ov ++++	+
A2569	+	+	—	Ov +	+
A2332	+	W+	—	Ov +	+
A2453	+	W+	—	Ov +	+
A2471	+w	+	—	Ov +	+

TABLE I—*Continued*

BIRD	TUBE	RAPID SERUM	WHOLE-BLOOD	GROSS	CULTURE
A2365	+f.	+	—	Ov ++	+
A2329	+gr.	+	—	Ov ++	+
A2422	+gr.	+	—	Ov +	+
A2543	+wGr.	+	—	Ov ++++	+
A2566	+w	+	—	Ov +	—
A2310	+	+	—	Normal	—
A2349	+	+	—	Normal	—
A2488	+	+	—	Normal	—
A2347	+	—	—	Ov ++	+
A2387	+	—	—	Ov +	+
A2407	+	—	—	Ov +	+
A2547	+	—	—	Ov +	+
A2213	+w	—	—	Ov +	+
A2320	+w	—	—	Ov +	+
A2444	+w	—	—	Ov +	+
A2350	+wf	—	—	Ov +	+
A2215	+gr.	—	—	Ov +	+
A2229	+gr.	—	—	Ov ++	+
A2462	+gr.	—	—	Ov +	+
A2548	+gr.	—	—	Ov +	+
A2316	+	—	—	Ov ++	—
A2497	+	—	—	Ov +	—
A2519	+w	—	—	Ov ++++	—
A2536	+gr.	—	— —	Ov ++	—
A2542	+gr.	—	—	Ov ++	—
A2613	+gr.	—	—	Ov +	—
A2392	+	—	—	—	+
A2380	+	—	—	—	—
A2307	+w	—	—	—	—
A2328	+gr.	—	—	—	—
A2406	+gr.w	—	—	—	—
A2304	—	+	—	Ov +	+
A2322	—	+	—	Ov ++	—
A2417	—	+	—	Ov +	—
A2459	—	+	—	Ov ++ Y	—
A2496	—	+	—	Ov +	—
A2541	—	+	—	Ov +	—
A2592	—	+	—	Ov ++	—
A2573	—	+	—	—	—
A2208	+	—	+	Ov ++	+
A2233	+	—	+	Ov ++++	+
A2397	+	—	+	Ov ++++	+
A2538	+	—	+	Yolk +	+
A2560	+	—	+	Ov ++	+
A2562	+	—	+	Ov ++++	+
A2230	w+	—	+s	Cyst	—
A2532	—	+	+	Ov +++	+
A2589	—	+	+	Ov +	—
No. 7	—	+	+	Ov +	—
A2345	—	—	+	Ov ++	+
A2348	—	—	+	Ov ++++	—
A2399	—	—	+	Ov ++++	—
A2514	—	—	+w	Ov ++	—*
A2545	—	—	+	Normal	—
A2552	—	—	+	Normal	—

*Staphylococcus.

postmortem for gross evidences of pullorum disease and cultures were made from unabsorbed yolks, diseased ova or any abnormality observed in any of the birds. Figure 1 and table I show the results and the correlation of the various tests with the bacteriological findings. Of the 94 birds which were positive to the tube agglutination method, 71 yielded cultures of *Salmonella pullorum*. Of the 75 birds which were positive to the rapid serum method, 54 yielded cultures of *S. pullorum*.

FIG. 1. Chart showing the number of birds detected by each of the taree methods, and the bacteriological findings at autopsy.

Of the 63 birds which were positive to the rapid whole-blood test (B. A. I. antigen), 47 yielded cultures of *S. pullorum*.

A study of this summary shows that 38 birds were positive to all 3 methods, showed gross lesions and yielded cultures of *S. pullorum* and that, of the 47 that showed a reaction to all three of the tests, 43 showed gross lesions of pullorum disease and 39 yielded cultures of this organism. In the group of 23

birds that were positive to the tube agglutination method alone, 18 showed gross lesions of pullorum disease and 13 showed cultures of *S. pullorum*. Of 8 birds that were positive to the rapid serum test and negative to the two other tests, 7 showed gross lesions and one yielded cultures of *S. pullorum*. Six birds that were positive to the whole-blood test and negative to the 2 other tests showed gross lesions in four birds and yielded cultures in one.

In culturing and studying lesions of this disease in positive birds, it was observed in some cases, that though marked gross lesions were present, the cultures that were made failed to yield *S. pullorum*. In a few cases where there were no evident lesions macroscopically, pure cultures of *S. pullorum* were obtained. Of the 23 birds which were positive to the tube agglutination test and not positive to the two rapid methods, 13 birds yielded cultures of *S. pullorum* (56.53 per cent). Of the 8 birds positive to the rapid serum test, one yielded cultures of *S. pullorum* (12.5 per cent). Of 6 birds that were positive to the whole-blood test, only one was positive bacteriologically (16.66 per cent). It is probable that if more organs had been cultured, a higher percentage of isolations would have been made. It is interesting to note, however, that a higher percentage of isolations was secured from the tube test than from either one of the other methods.

It can be seen from the chart (fig. 1) and the data given that the rapid serum and the whole-blood tests are not so efficient in detecting the disease in flocks in our hands as the tube agglutination test, and that the rapid serum test is better than the whole-blood test.

The bacteriological findings in our investigation show that not only will the tube method pick out a great many more birds, but that in all cases the tube test is more reliable.

The better results obtained with the tube test may, in part, be explained on the basis of our method of reading the test. It is the practice in our laboratory to regard as a positive reaction any changes from the normal cloudiness that may occur in a tube. In some experiments to determine whether these slight changes that occur in the tube had any relation to the disease, twelve birds were purchased, which gave a so-called granular reaction or a reaction which some laboratories have regarded as a negative reaction to pullorum disease, and careful post-mortem and bacteriological examinations of the birds were

made. Eight of the 12 examined were positive culturally for pullorum disease.

In the data submitted in this paper, 12 of the 94 birds which were positive to the tube method showed slight reactions, or so-called granular reactions, and seven of these birds yielded cultures of *S. pullorum.* Moreover, three of the five remaining birds that were negative culturally showed lesions of the disease.

In cleaning up flocks in Missouri, we feel that a careful reading of the tests increases the efficiency in removing infected fowls from a flock and therefore more quickly eliminates the infection.

In conclusion, the author wishes to suggest that until the rapid tests have been proven as accurate and dependable as the tube agglutination test, they should not be substituted in campaigns for the eradication of pullorum disease. One state, at least, which has substituted the rapid serum test for the tube test, is convinced that this test is as dependable and reliable as the tube agglutination test. When the rapid tests have proven themselves as good as the old tube test, it is then, and only then that they should be recommended as a means of eradicating pullorum disease from farm flocks. It is hoped that a more simplified and practical method will be developed that does not involve the mechanics that are required for the tube agglutination test.

The presentation of this paper, therefore, is not in a form of criticism of any test, but a piece of investigational work designed to help establish some definite line of investigation that will finally prove beyond doubt what test is the best and most practical for the eradication of pullorum disease.

Summary

1. In a single test on a flock of 259 birds to determine the relative efficiency of the tube agglutination test, the rapid serum test and the whole-blood test (using a stained antigen), the results indicate that the tube test is more efficient than either the rapid serum or the whole-blood test.

2. The rapid serum test is more efficient in our hands than the whole-blood test.

3. More than twice the number of isolations of the organism were secured from birds positive to the tube test and negative to the other tests.

4. The reading or interpretation of the test may partly account for the better results obtained with the tube test in our hands than the two other methods.

5. Any change from a normal cloudiness in a tube or a so-called granular reaction is regarded as a positive reaction in this laboratory.

6. Of 24 birds which showed the slight reaction or so-called granular reaction, 15 were positive culturally for pullorum disease and three of the nine remaining birds that were negative culturally showed lesions of the disease.

ACKNOWLEDGMENTS

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Record Made in Eradicating Foot-and-Mouth Disease

Effective control of the recent outbreak of foot-and-mouth disease in southern California, with prospects that the plague probably had been eradicated, was announced by the U. S. Department of Agriculture, early in June. The original outbreak was diagnosed on April 28, and the last infected herd was slaughtered and buried May 7. No other signs of foot-and-mouth disease, either in the quarantined area or surrounding territory, had been found up to a recent date. All infected premises had been thoroughly cleaned and disinfected, and restocking was begun June 15. Although many of the veterinarians assigned to the task of eradicating the outbreak have now returned to their regular official stations, a sufficient force has been left in the quarantined area to handle any emergency that may arise.

Eradicating the disease in a period of ten days sets a new record and is noteworthy in comparison with former outbreaks, one of which required 18 months for eradication. The shortest previous time which elapsed between the diagnosis of the disease and the disposal of the last infected herd was 31 days.

SPONTANEOUS INFECTION WITH BRUCELLA ABORTUS IN THE BULL*

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For many years the question has been debated as to whether bulls from abortion-infected herds, more especially bulls that react to the serologic tests for *Brucella abortus*, are capable of transmitting the infection to cattle in clean or non-reacting herds. The problem has both scientific and practical implications, so any further light that can be shed upon it should be of interest to all persons concerned with Bang's disease control.

While some studies have been made with bulls both naturally and artificially infected with *Br. abortus*, few or no carefully controlled experiments on spontaneously infected bulls that were blood-tested over a considerable period, bred to virgin heifers, castrated, slaughtered, and their semen and tissues studied bacteriologically, have been reported.

When an opportunity was given to conduct such a study, advantage was taken of it, as it was felt that the results would help answer the question propounded above and furnish more information relative to the management of both clean and infected herds.

The first recorded attempt to infect clean cows by breeding them to an abortion-infected bull that the authors have been able to find in the literature is that of M'Fadyean and Stockman.[1] They failed to establish the infection in any of their test cows.

Hadley and Lothe[2] reported that they were unable to infect non-reacting, virgin heifers by mating them with reacting bulls. They accounted for this by attributing to the bull greater ability to overcome infection with *Br. abortus* than is possessed by cows and heifers. Their results were similar to those secured by the English workers mentioned above, and led them to conclude that the bull plays little or no part in the dissemination of contagious abortion. No cultural studies were made of the genitalia, so the experiment was not adequately controlled.

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Missouri, August 25-28, 1931. Published with the approval of the Director of the Wisconsin Agricultural Experiment Station.

Buck, Creech and Ladson[3] studied four naturally infected, reacting bulls and succeeded in isolating *Br. abortus* from the testicle of one and the seminal vesicles of the others. They did not attempt to infect cows by using the infected bulls for service. They concluded that the seminal vesicles furnish the most favorable site for the lodgment and propagation of *Br. abortus*. Their work led them to state that the organism appears to be more strongly indicated by relatively marked reactions than by slight reactions to the agglutination test.

Lubbehusen and Fitch[4] tried to establish active testicle infection in four bulls by injecting *Br. abortus* deep into the testicular tissue. Although all of the test animals became well-marked reactors to the agglutination test, it was impossible to prove the presence of the organism in the semen of any of them at any time.

Carpenter and Boak[5] state that the bull is not so susceptible to infection with *Br. abortus* as is the cow. This statement is supported by nearly all other students of Bang's disease. That the bull does become actively infected was demonstrated by Carpenter,[6] who recovered the organism from the semen of two reacting bulls and from the epididymes or seminal vesicles of four infertile bulls.

Connaway[7] sums up the opinion at present most widely held in the following statement:

> Bulls may become infected in the same way that cows become infected; that is, by eating materials soiled by the infected uterine discharges. The bull that runs free with an infected herd is more likely to contract the disease than one that is kept in a separate pen during the intervals of breeding. A bull that is infected with the Bang abortion germs may become sterile from disease of the testicles, and may also transmit the infection to cows. Transmission of the disease by a healthy bull, in a mechanical way during breeding, occurs but seldom and can easily be prevented.

The percentage of bulls that show the presence of *Br. abortus* agglutinins in their blood serum has been computed by many investigators, but seems to be no index to the individuals that have active lesions in the genital organs. There is no doubt, however, that the bull is much less susceptible to infection than is the heifer and the cow. This also applies to the male of other species of farm animals as compared with the females.

From the above and other reports it seems to be the consensus of opinion that while the bull may be a passive carrier of the infection, he seldom becomes actively infected and even then is not likely to serve as a spreader.

OBJECTS OF THE EXPERIMENT

The purposes of the present work have been:

1. To determine if *Br. abortus* could be isolated from the semen of a bull with a spontaneous infection.

2. To learn whether such a bull could infect virgin, non-reacting heifers through service and cohabitation.

3. To determine whether or not the agglutination titre of the bull's blood would be affected by the removal of the infected testicle.

4. To attempt to recover *Br. abortus* from the testicles when they were removed.

HISTORY OF THE TEST BULL

The following letter from the breeder of the bull records the history of the animal so well that it is quoted verbatim:

———, Wis., April 6, 1930.

I am sending herewith another blood sample from the young bull as per your request. I was very much surprised to learn that he is a reactor. He was bred to two cows on February 21 and March 13 respectively. Both of these animals at time of service were reactors to the blood test. I will give you a bit of history, as it may assist you in making your deductions.

About New Year's time we noticed a lameness in his right hind leg. At first we thought it some slight injury, but we saw it was getting worse. On examination we discovered that that testicle was much enlarged. It got to be about three times its normal size. We treated him and it reduced. And at the time we bred the first cow February 21 it had not fully come down to normal size. It did, however, in a week or more afterward and I thought he was all right. A day before I sent in the last blood sample a man was here looking for a bull. He wanted him tested and I told him that I was sending in the blood of my herd on the morrow. His reaction as reported by you on March 21 caused me to make further examination. I find that testicle to be considerably reduced below normal in size and is considerably hardened. It is apparent now that he has only one live testicle, and whether his seed is fertile, I do not know. I do not know that he was injured. Perhaps this disease caused this disturbance. I am wondering if the disease is seated in this testicle. His dam is a non-reactor.

This bull is small for his age. Somehow I was never able to get him to grow as other calves usually do here at Guernseyville.

In a later letter the owner reported that neither of the reactor cows, mentioned above as having been bred to this bull, conceived to his service.

From the above description it is quite evident that the bull became infected with *Br. abortus* during the latter part of 1929. This was before he was used for service, so that it is probable that the organisms gained entrance to the body by way of the digestive tract, or possibly through the conjunctiva, rather than by the uro-genital tract. This assumption is supported by the fact that the bull had always been fed skim milk from a herd in which

more than fifty per cent of the animals were known to be reactors to the agglutination test for Bang's disease, and in which thirty per cent of the cows had actually aborted.

With such a complete history we realized that this animal would serve admirably for experimental work. Accordingly we purchased him and brought him to the Wisconsin Agricultural Experiment Station at Madison, early in April, 1930, where he was kept under close observation for exactly six months. He was placed at the head of the herd, consisting of nine test heifers, to be described later.

Experimental Procedure

In order to secure a complete picture of the bull's blood serum reactions to the agglutination test for abortion, blood samples were drawn once a month or oftener. These tests were made by the rapid or plate method, and the results, recorded in table I, show that, before the surgical removal of the testicle, no significant decrease occurred in the titre of the blood. Within 16 days after the operation the titre had decreased by one-half, and in less than 100 days had become negative.

Table I—*Agglutination reactions of the test bull (rapid method test).*

Date (1930)	Amounts of Serum and Results of Tests			
	.02	.01	.005	.002
March 21	+	+	+	+
April 1	+	+	+	P
April 12	+	+	+	P
April 28	+	+	+	P
May 12	+	+	+	P
May 28*	+	+	+	P
June 13	+	+	P	—
July 9	+	+	S	—
July 28	+	S	—	—
Sept. 3	—	—	—	—
Oct. 1	—	—	—	—

*Right testicle surgically removed.

On April 29, 1930, the bull was bred to one of the virgin heifers in the test herd. A sample of the semen was recovered from the vagina and 1 cc was injected intraperitoneally into a series of six guinea pigs, with the result that two of the guinea pigs died within a few days; two did not develop agglutinins specific for *Br. abortus* and failed to show lesions of brucelliasis at autopsy; one became a four-plus reactor and had an enlarged spleen witn necrotic areas, from which the organism was isolated

by culture under reduced oxygen tension; and the control remained negative. These results are presented in table II.

TABLE II—*Guinea pigs injected with semen.*

EAR-TAG	INJECTED			FATE	SERUM REACTION				LESIONS
	DATE	SEMEN (CC)	METHOD		.02	.01	.005	.002	
C220.1				D. 5–1					
C553.1				D. 5–3					
C37	Apr. 29	1.0	Intraperi-	K. 6–16	—	—	—	—	None
C575.2			toneal	K. 6–16	+	+	+	+	Note 1
C690.2				K. 6–16	—	—	—	—	None
C644.2	Control			K. 5–16	—	—	—	—	None

Note 1 = spleen enlarged; *Br. abortus* recovered.

As indicated above, the bull was turned into a paddock with nine test heifers on April 29. The cattle were allowed to run together continuously until October 8, 1930, when the bull was removed and slaughtered. It was impossible under this system of management to determine the exact dates of service. However, it is certain that each of the heifers conceived promptly to service by him, as all were found to be heavily pregnant when they were slaughtered on January 8, 1931.

On May 28, 1930, after the bull had served most, if not all, of the heifers, his right testicle was surgically removed. The organ on inspection was smaller and firmer than normal—indicating a previous acute inflammation. A series of four guinea pigs were inoculated with an emulsion of the testicle. Three of these pigs died within a few days. The fourth was kept until July 23, when it was killed. Its blood serum was negative to the test for abortion in all dilutions. No lesions were evident in the spleen or other internal organs. It is likely that the testicle at time of removal was not infected with *Br. abortus*, but this is not proved because three-fourths of the test pigs died before they could have developed a specific reaction.

The heifers were blood-tested on April 28, May 28, June 18, July 9 and 28, September 3, October 2 and December 31. No trace of a reaction was given, even in the lowest dilution of serum (1:50), at any time. This indicates that the heifers did not contract Brucella infection from the bull through cohabitation, although he was a marked reactor at the time he bred them and was discharging *Br. abortus* in his semen at least during part of the period they were kept together.

It was necessary for economic reasons to slaughter the heifers on January 8, 1931. At this time all of them were found to be well advanced in pregnancy. There was no evidence of Brucella infection in the cotyledons or in the utero-chorionic space. With the exception of a small abscess in the region of the right humerus of heifer 11 and skin lesions of no significance in the region of the right and left scapulae of heifer 12, nothing abnormal was noted in any of the nine animals.

The bull was slaughtered October 8, 1930, and the left testicle was secured for study. Gross inspection of all genital organs failed to reveal pathological changes, so only the testicle was cultured for *Br. abortus*. The results were negative; in other words, no evidence of infection with this organism was demonstrated by the cultural method. An emulsion of the testicular tissue was injected into two guinea pigs. The latter were killed by bleeding, December 1, 1930, but their glandular organs appeared to be normal and their blood sera were negative to the agglutination test.

History of an Interesting Contemporary Bull

Another bull that sheds some light on this problem is the one which has been for four and one-half years at the head of the nutrition-abortion experiment herd at the University of Wisconsin. This herd originally consisted of 44 negative heifers, all of which, after dropping normal calves, were rebred and at about the 76th day of pregnancy were exposed to *Br. abortus* infection. A description of the experiment and the results may be found in a paper by Hadley and Hawn,[8] and in two annual reports of the Director of the Wisconsin Agricultural Experiment Station[9]. Suffice it to state that more than one-half of these cows aborted, and that although this bull headed the herd during the entire period, his blood serum never gave even the slightest suggestion of a reaction. Moreover, his breeding efficiency was in no way affected, for most of the cows conceived to service by him after the storm of induced abortion swept through the herd.

Discussion

The evidence presented clearly shows that the test bull discharged *Br. abortus* in his semen. Whether the microörganisms came directly from foci of infection in the testicle or from foci in some other organ of the uro-genital system has not been proved. As a matter of fact they may have come from either

source, because we failed to demonstrate *Br. abortus* in either of the testicles and did not make a bacteriologic examination of the accessory organs of reproduction at time of slaughter.

It is a well-established fact that the functional activity of acutely inflamed glands, in some cases, is completely inhibited, while in other cases a pathologic secretion is produced in which the infecting organisms are carried. Whether the acute orchitis in our bull was the direct result of invasion of the testicle by *Br. abortus* alone or in combination with some other disease-producing organism will never be known. In fact, as intimated above, and because so many of the guinea pigs died within a few days after inoculation, there is a possibility that our test bull did not have a testicular infection with *Br. abortus*. However, the probability is that he did.

The evidence is clear that the heifers to which the bull was bred failed to become infected. This fact tends to indicate that a bull with an active genital infection with *Br. abortus* may not infect susceptible heifers, even though he is discharging the organism in the semen. It also indicates that heifers are not readily infected by way of the vagina.

On first thought it seems odd that the heifers did not become infected. That this might be expected is shown by the work of Schroeder and Cotton[10] who found that abortion bacilli, after injection into the non-pregnant uterus of a cow, disappeared within a few days. The results secured by Giltner and Bandeen[11] who failed to isolate the organisms, when introduced into the vagina or sheath, in most cases after twenty-four hours, also helps to explain our results. Consequently it seems that when *Br. abortus* is introduced at time of service, there is little likelihood that an infection will be established.

CONCLUSIONS

This experiment is another attempt to accumulate more evidence upon a moot question. The results are not clear-cut enough to justify drawing final conclusions. However, it seems reasonable to conclude from our results and those of others who have studied Brucella infections that:

1. Testicle infection with *Brucella abortus* occurs in the bull from having acquired the organism by natural contacts.

2. Bulls discharging *Brucella abortus* in their semen are not necessarily capable of infecting susceptible cows which they serve.

3. Bulls may be used for years in badly infected herds, yet not acquire the infection.

4. The fact that a bull reacts to the blood test for abortion infection is not conclusive evidence that *Brucella abortus* is being eliminated in his semen.

5. The higher the agglutination titre in a bull, the greater the possibility of his having an active infection in some organ of the uro-genital system.

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Progress in Eradicating Avian Tuberculosis

A summary of progress in eradicating tuberculosis from poultry is now being issued monthly by the U. S. Bureau of Animal Industry. The summary includes the results of inspection of poultry flocks in eleven states where systematic work in detecting and eradicating tuberculosis of poultry is under way. These states are: Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, Ohio, South Dakota and Wisconsin. In addition, there are reports for ten other states in which veterinarians engaged in testing cattle for the disease also inspected poultry. The report includes the tabulated results of post-mortem examinations of flocks and fowls affected with tuberculosis, as shown by clinical examination or the tuberculin test. The summary for February showed 8,695 flocks under supervision for the eradication of avian tuberculosis.

On to Atlanta
August 23-24-25-26

EXPERIENCES IN ERADICATING BANG'S DISEASE IN THREE INFECTED HERDS OF CATTLE*

By C. F. CLARK, *East Lansing, Mich.*

Michigan Agricultural Experiment Station

The purpose of this paper is to relate some experiences in attempting to eradicate Bang's disease, and to discuss the significance of low agglutination titres and the frequency of abortions in negative animals. The three herds of cattle studied have been under the close observation of members of the Department of Animal Pathology for several years. Bang's disease has existed in each of these herds, though to a varying degree.

In each of the herds studied, the method used in attempting to eradicate the disease was the frequent application of the agglutination test and the subsequent segregation of positive animals. Vaccination as a control measure was not resorted to, as it was desired to test the feasibility of the first-mentioned plan. Each of the three herds is frequently visited by farmers, cattle-buyers and others. In close proximity to one of the herds (C) until the fall of 1930 was a Brucella-infected herd of swine. All three of the herds are within one-half mile of a Bang-infected herd of cattle, although there is no opportunity for contact between the animals. There is a possibility of infective material being transferred on the feet of humans, birds, dogs and cats.

The method used in testing the blood sera of the herds is the Huddleson and Abell[1] rapid-plate method. The dilutions used correspond to dilutions of 1:25, 1:50, 1:100, 1:200 and 1:500 used in the test-tube method. A serum showing a trace or incomplete reaction in 1:25 was classed as negative. Serum titres, ranging from + in 1:25 to + in 1:50 were classed as suspicious. A positive serum titre of 1:100 or above was considered as evidencing infection.

HERD A

This herd comprises about 60 head of dairy cattle. During the period of observation it has consisted principally of young cows and heifers. Bang's disease existed in this herd for several years, although its spread had been markedly reduced by temporary segregation of cows at calving time and feeding in individual

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mangers. At the time this study was begun, there was one positive animal in the herd. During the first year of observation, monthly agglutination tests were conducted on all animals of breeding age, and the policy of temporary isolation at parturition was continued. In the fall of 1930, new quarters were ready for occupancy. As the serum titre of the one positive cow (see table I) had become lower, finally entering the negative class, all animals were taken into the new quarters. Since that time, tests have been run at approximately sixty-day intervals.

Table I summarizes the breeding history of herd A.

TABLE I—*Summary of breeding history of herd A, July 1, 1929 to July 1, 1931 (Animals grouped according to maximum agglutination titre).*

		NEGATIVE	TRACE IN 1:25	PARTIAL IN 1:25	POSITIVE IN 1:25	POSITIVE IN 1:500	TOTAL
Heifers		2	1	1	0	0	4
Cows		17	11	16	5	1	50
Parturitions		24	13	22	6	2	67
Parturitions terminating in abortions	No.	2	1	0	0	0	3
	%	8.3	7.6	0	0	0	4.4
Retained fetal membranes	No.	3	0	4	3	0	10
	%	12.5	0	18.1	50.0	0	14.9

TABLE Ia—*Abortions in herd A.*

ANIMAL	MAXIMUM AGG. TITRE	BRED	ABORTED	GESTATION PERIOD (DAYS)	CULTURAL FINDINGS
G8	Negative	1-21-29	9-10-29	232	Not cultured
G1	Negative	6-24-30	2-28-31	249	*B. coli* recovered from fetal organs
G20	Trace in 1:25	12-31-30	4-28-31	118	*B. coli* recovered from fetal organs

Discussion of table I: It is apparent that 50 cows should deliver more than 67 calves in a two-year period. This may be explained by stating that 12 of the animals recorded delivered their first calves during the second year of the period studied. Fifteen animals have left the herd, 2 by death and 13 sold for slaughter after calving once during the period studied.

Each of the 5 cows listed as positive in 1:25 has a normal breeding history. One is a heifer calving the first time in February, 1931; 1 is a cow calving twice; the other 3 cows were sold for slaughter, having calved once each during the period studied.

Each of these cows retained the fetal membranes after calving. Table Ia is self-explanatory.

Attention is directed to the agglutination record of one cow, G10. (See table Ib.) This animal has apparently entered the negative group permanently. She has a normal breeding history, delivering normal healthy calves, May 26, 1930, and April 30, 1931.

TABLE Ib—*Agglutination record of cow G10.*

	JAN.	FEB.	MAR.	APR.	MAY	JUNE
1929						
1930	+P P T −	+P T − −	P P T − −	P T T − −	T − − − −	P T − − −
1931	T T − − −		P T − − −		P T − − −	

	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
1929	+ +P T −	+P T − −	+ + +P −	+ + + +T	+ +P T −	+P T − −
1930	P T − − −	+P T − −	T T − − −		P T − − −	
1931	P T − − −					

− = absence of.
T = trace of.
P = partial or incomplete.
\+ = complete agglutination.

HERD B

Herd B is composed of dairy cattle of all ages. Bang infection existed in this herd for several years in a very virulent form. The agglutination test had been applied frequently and segregation practiced. The positive cows were kept on one side of the barn and pastured in separate lots. In July, 1929, 24 of 98 head (25 per cent) tested were positive. During June and July, 1929, 22 new animals were assembled from several different herds and stabled in a new building. These animals, of course, had been

TABLE II—*Summary of breeding history of herd B, July 1, 1929 to July 1, 1931 (Animals grouped according to maximum agglutination titre).*

		NEGATIVE	TRACE IN 1:25	PARTIAL IN 1:25	POSITIVE IN 1:25	TOTAL
Heifers		29	7	5	0	41
Cows		40	26	21	5	92
Parturitions		62	40	34	7	143
Parturitions terminating in abortions	No.	5	2	5	0	12
	%	8.1	5.0	14.7	0	8.3
Retained fetal membranes	No.	8	9	6	1	24
	%	12.9	22.5	17.6	14.2	16.7

previously tested and found negative. In July, 1929, all negative cows were removed to the new buildings. The negative heifers had previously been turned out to pasture.

The 24 positive cows remained in the old barn and were sold for slaughter as they became unprofitable. It is of interest to note that 14 of the 24 (58 per cent) had aborted at some period in their breeding history.

From July, 1929, to July, 1930, monthly agglutination tests were run, since then at 60-day intervals.

Table II summarizes the breeding history of herd B.

TABLE IIa—*Abortions in herd B.*

ANIMAL	MAXIMUM AGG. TITRE	BRED	ABORTED	GESTATION PERIOD (DAYS)	CULTURAL FINDINGS
65	Partial in 1:25	12-23-28	8-13-29	233	*B coli* recovered from all fetal organs
226	Partial in 1:25	6-18-29	11-12-29	147	Twins. Developmental anomaly of fetal membranes
251	Trace in 1:25	5-20-29	11-14-29	178	Vibrio recovered from fetal liver
241	Negative	8- 6-29	1-17-30	164	Not cultured. Mummified
67	Negative	11-25-29	2-20-30	87	Cow had severe case of pneumonia
65	Partial in 1:25	11-24-29	6-17-30	207	Vibrio recovered from fetal organs
6	Partial in 1:25	3- 6-30	8- 8-30	154	Not cultured
9	Trace in 1:25	4-28-30	12- 9-30	225	*Br. abortus* cultured from fetal organs
83	Negative	6-22-30	1- 4-31	196	Cultures negative
229	Partial in 1:25	8- 5-30	3- 7-31	214	Twins. Cultures negative
78	Negative	8-16-30	3-24-31	220	Cultures negative
169	Negative	2-19-31	6-18-31	119	Fetus not recovered

Discussion of table II: It is evident that 92 cows should produce more than 143 calves in a two-year period. Among the parturitions listed were those of 22 heifers calving for the first time in the second year of the period studied. Several animals were sold as pregnant cows, 3 died, 4 were sold for slaughter.

The 5 cows listed in the column headed positive in 1:25 represent 2 heifers calving for the first time, one cow calving twice, and 2 pregnant July 1, 1930.

Table IIa summarizes the abortions occurring in herd B during this two-year period. It will be seen that the apparent cause was

ascertained in 6 of the 12 cases, in 3 cases the fetuses were not cultured, and in the remaining 3 cases the cultural findings were negative.

Of especial interest is the cow No. 9. This animal was purchased as a pregnant negative cow in July, 1929, calved normally December 7, 1929, was bred February 9, 1930, March 1, 1930, March 23, 1930, and April 28, 1930, conceived to the latter date, and aborted December 9, 1930. The fetal membranes were retained, and the cow developed a fatal septicemia. This cow has 13 agglutination tests on record, trace in 1:25 in August, 1929, and in August, 1930, and the remaining 11 tests negative. The only known history of exposure is that this animal was bred to a bull which had a low agglutination titre. It is very unfortunate that this animal died, as it would have been interesting to know if a positive reaction would have developed after the abortion.

While cultural findings on three of the last four abortions were negative, the necropsy picture was strikingly similar. Very large amounts of a sanguineous fluid were present in the body cavities of the aborted fetuses.

The abortion rate of 8.1 per cent in herd B appears to be, and is, a high rate for presumably negative animals. Other workers have reported rates in certain negative herds comparable with this. Rettger, McAlpine, et al[2] report an abortion rate of 6.2 per cent on a negative herd. Palmer[3] reporting on several herds, found annual abortion rates in certain herds of 9.3 per cent, 9.7 per cent, 10.3 per cent, and 11 per cent. Huddleson and Smith[4] found an abortion rate of 15.4 per cent for negative animals. Some of these cows had associated with positive animals, which may modify its value.

It should be remembered that although many of the cows in herd B were drafted from the old infected herd, no positive reactors have developed.

Herd C

This herd is composed of beef cattle of all ages. Bang's dsiease existed in this herd for several years. The incidence of the disease had been reduced by selling for slaughter some of the cows which did abort, and by segregating at parturition. This method was not entirely successful, as 17 per cent of the herd reacted on the first test conducted during the period reported.

Several tests were made, and in the spring of 1930 the positive and negative cattle were turned out for the season into separate

pastures. In the fall of 1930 only the negative and a few suspicious animals were put into the barn. At this time there were 9 positive cows remaining. They were completely isolated from the negative group and eventually all sold for slaughter. Since the negative herd has been established, 7 tests have been conducted on all animals over 8 months old. No positive reactions have developed.

Tables III and IIIa summarize the breeding history of herd C.

TABLE III—*Summary of breeding history of herd C, July 1, 1929 to July 1, 1931 (Negative animals grouped according to maximum agglutination titre).*

	NEGATIVE	TRACE IN 1:25	PARTIAL IN 1:25	POSITIVE IN 1:25	POSITIVE IN 1:50	TOTAL
Heifers	29	6	5	2	0	42
Cows	14	8	10	5	1	38
Parturitions	20	14	14	9	3	60
Parturitions terminating in abortions	0	0	0	0	0	0

TABLE IIIa—*Positive animals in herd C.*

Animals		17*
Parturitions		22
Parturitions terminating in abortions	No.	2
	%	9.1

*Eight of these 17 positive cows had histories of having aborted previous to July 1, 1929; therefore, out of 17 positive cows, 10 (58.8 per cent) had histories of having aborted.

Discussion of table III: It is obvious that 38 cows should deliver more than 60 calves in a two-year period. There were several heifers calving for the first time included in this group; a few cows were sold for slaughter after calving once during this period. Each mature cow has delivered 2 normal calves during this period, with one exception. The cow excepted, listed as positive in 1:50 in table III, calved 3 times.

Attention is directed to the 7 animals listed in the column headed positive in 1:25. The 2 classed as heifers represent unbred heifers. One of these 2 was slaughtered in April, 1931. Cultures of the udder and genitalia were negative for *Br. abortus*. The 5 cows all have normal breeding histories; 1 was sold for slaughter, after calving once, and the remaining 4 have delivered 2 calves each.

Discussion of tables IV and V: In considering table IV, it will be observed that the abortion rate for the three first groups did not vary greatly. This would suggest that the trace and incomplete reactions on these animals were caused by non-specific agglutinins.

Although the number is too small on which to base conclusions, it is interesting that none of the animals having a positive titre of 1:25 have aborted. Possibly these reactions were caused by non-specific agglutinins.

TABLE IV—*Summary of breeding records of herds A, B, and C, July 1, 1929 to July 1, 1931 (Animals grouped according to maximum agglutination titre).*

		NEGATIVE	TRACE IN 1:25	PARTIAL IN 1:25	POSITIVE IN 1:25	POSITIVE IN 1:50	POSITIVE IN 1:500	TOTAL
Animals		131	59	58	17	1	1	267
Heifers		60	14	11	2	0	0	87
Cows		71	45	47	15	1	1	180
Parturitions		106	67	71	21	3	2	270
Parturitions terminating in abortions	No.	7	3	5	0	0	0	15
	%	6.6	4.4	7.0	0	0	0	5.5

TABLE V—*Summary of 3021 agglutination tests on blood sera of animals in herds A, B, and C, July 1, 1929 to July 1, 1931 (Animals grouped according to maximum agglutination titre).*

HERD	NEGATIVE	TRACE IN 1:25	PARTIAL IN 1:25	POSITIVE IN 1:25	POSITIVE IN 1:50	POSITIVE IN 1:500	TOTAL
A	19	12	17	5	0	1	54
B	69	33	26	5	0	0	133
C	43	14	15	7	1	0	80
Total	131	59	58	17	1	1	267
Per cent	49.1	22.1	21.7	6.3	0.4	0.4	100.0

Birch and Gilman[5] state that "in non-infected cows the agglutination reaction fluctuates in the dilutions up to, and including, 1:40. This is due to normal, or group, agglutinins."

Derrick[6] states that "normal animals will often give a complete agglutination at 1:20 and occasionally at 1:40."

Table V summarizes the data on agglutination tests conducted on all animals tested in the three herds. Of the 267 animals tested, 248 (93 per cent) never reacted higher than incomplete in 1:25. Seventeen (6 per cent) had a maximum serum titre of positive in 1:25.

Conclusions

In the three herds under observation, Bang's disease has apparently been eradicated by frequent application of the agglutination test and permanent segregation of positively reacting animals.

The abortion rate of animals having a maximum agglutination titre of trace or incomplete in 1:25 did not vary appreciably from those negative.

In the three herds studied, the abortion rate varied from 0 to 8.3 per cent with a mean of 5.5 per cent.

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[4]Huddleson, I. F., and Smith, L. H.: A critical study of the Brucella agglutination reaction and abortion rate in a herd of cattle under natural conditions. Jour. A.V.M.A., lxxix (1931), n. s. 32 (1), p. 63.

[5]Birch, R. R., and Gilman, H. L.: The agglutination test as an aid in handling Bang abortion disease. Corn. Vet., xvi (1926), 1, p. 127.

[6]Derrick, G. W.: A report on the application of a plan for the control of Bang abortion disease. An. Rpt. N. Y. State Vet. Coll. at Corn. Univ., 1927-1928, p. 102.

Arizona-New Mexico Meeting Called Off

The joint meeting of the New Mexico and Arizona State Veterinary Medical Associations, which was announced in the Journal (May, 1932, p. 812), was not held, due to the fact that so many federal and state veterinarians were called to California in connection with the recent outbreak of foot-and-mouth disease.

Kellogg Horse Ranch Given to University of California

Newly-acquired possession of the University of California is the W. K. Kellogg Arabian Horse Ranch at Pomona, Calif., recently presented to the University by W. K. Kellogg. The gift includes a 750-acre ranch, 87 blooded Arabian horses and a $600,000 endowment. The Kellogg stud has raised some of the finest Arabian horses in the United States, and much has been done there to improve the strain of western saddle horses.

Many of the A. V. M. A. members who attended the Los Angeles convention in 1930 had the pleasure of visiting the Kellogg Ranch and seeing the splendid array of Arabian horseflesh brought together by Mr. Kellogg.

The Fourth International Agricultural Education Congress will convene in Rome, Italy, in November, 1932.

A CLINICAL STUDY OF FORTY CASES OF DISEASE OF THE REPRODUCTIVE ORGANS OF THE COW*

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During the college years of 1927 to 1929, inclusive, the author had an opportunity to make a clinical study of a limited number of cases of disease of the reproductive organs of cattle. Because of a change in position, it will be impossible to continue this type of work, so it is desirable to present this material at this time.

The accompanying table presents clinical records of forty cases of diseased genital organs of the cow. Thirty-four of these were treated for sterility, nine of which were treated also for retained fetal membranes, and six cases were treated for retained fetal membranes only. All animals treated during the above-mentioned period are included, regardless of the results of the treatment. Since the data are presented in a rather complete and detailed form, little explanation or discussion is necessary.

By way of explanation for the variation in concentration of Lugol's solution used for douching, it might be said that the severity of the condition was the deciding factor. If the case to be treated showed indications of being only a slight catarrhal metritis, a weaker solution was used. However, when a severe condition, such as chronic metritis or pyometra was to be treated, Lugol's solution as strong as 4 or 5 per cent constituted the douche.

In treatment of retained fetal membranes, mineral oil and iodoform seemed to be the most reliable when properly administered. However, the 0.7 per cent pepsin solution containing 0.5 per cent hydrochloric acid gave very satisfactory results, in the few cases treated with it, in digesting and loosening the placental areas. This method of treatment appears to be worthy of more study in view of the fact that, in laboratory experiments conducted by the author, parts of fetal membranes placed in a solution of 0.7 per cent pepsin were digested to the extent of 91 per cent within 48 hours.

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†Resigned July 1, 1929.

TABLE I—*Clinical records of forty cases of diseased genital organs of the cow.*

Previous to Treatment					Period of Treatment			Subsequent to Treatment				
Cow and Date of Birth	Reaction to Ago. Test for Br. Abortus	Termination, Length of Gestation and Parturition Number*	Sequelae	Services Following Last Parturition	Diagnosis of Condition	Date	Treatment	Services	Conception (Date)	Termination and Length of Gestation	Condition of Calf	Remarks
44 7-27-19	+	1-24-27 267 da. (5)	Calf (N)	4	Catarrhal metritis and cervicitis	10-11-27	Douched uterus with 2% Lugol's solution, swabbed cervix with concentrated Lugol's sol.	1	11-1-27	7-26-28 267 da.	Normal 86 lbs.	Calved normally to first service
138 4-23-21	—	12-6-27 279 da (5)	Calf (N)		Retained after-birth	12-8-27 12-10-27 12-12-27	Injected minera oil and iodoform Injected mineral oil and iodoform Fetal membranes removed	4	10-1-28	7-15-29		
					Metritis	3-19-28	Douched uterus with 2% Lugol's sol.			287 da.	Normal	
141 7-8-21	—	11-20-26 252 da. (4)	Retained after-birth Twins (D)	3	Catarrhal metritis	7-27-27	Douched uterus with 4% Lugol's sol.	2	9-11-28	6-19-28 281 da.	Normal	
		6-19-28 281 da. (5)	Calf (N)	2	Cystic ovaries	10-4-28	Cysts crushed at six different times	11				Not pregnant
187 1-7-24	+	11-14-27 222 da. (3)	Calf (D)		Retained after-birth	11-15-27 11-18-27 11-20-27	Injected mineral oil and iodoform Injected mineral oil and iodoform After-birth removed	1	5-5-28	2-8-29 279 da.	Normal	Pregnant again to second service
					Metritis, lack of ovarian activity	3-20-28	Uterus douched with 2% Lugol's sol.					
188 1-13-25	—	2-7-28 277 da. (3)	Calf (N)		Retained after-birth Cystic c. l.	2-8-28 2-13-28 3-19-28	Injected 1000 cc 0.7% pepsin sol. After-birth removed Expressed cystic c. l. from left ovary	2	6-7-28	3-14-29 279 da.	Normal. 75 lbs. Female	Pregnant again to first service

TABLE I—*Continued.*

Previous to Treatment					Period of Treatment			Subsequent to Treatment				
Cow and Date of Birth	Reaction to Agg. Test for Br. Abortus	Termination, Length of Gestation and Parturition Number*	Sequelae	Services Following Last Parturition	Diagnosis of Condition	Date	Treatment	Services	Conception (Date)	Termination and Length of Gestation	Condition of Calf	Remarks
209 3-7-24	+	4-27-28 241 da. (3) 2-23-29 257 da. (4)	Calf (D) Male (N) Female (D)		Retained after-birth Retained after-birth	4-27-28 5-1-28 2-25-29 2-27-29	Injected mineral oil and iodoform Removed after-birth Injected 1000 cc 0.7% pepsin sol. Removed after-birth	1	6-30-28	2-23-29 257 da.	Male Appeared healthy Female (D)	Slaughtered 9-19-29 Poor individual Apparently normal upon autopsy
217 4-20-25	—	7-29-28 275 da. (3)	Calf (N)	5	Catarrhal metritis	3-27-29	Douched with 0.85% salt sol.	2				Slaughtered 5-24-29 Was pregnant to a service 22 days following treatment
230 9-3-25	—	9-23-28 270 da. (2)	Calf (N)		Disturbed ovarian function. Granular vaginitis	12-28-28	Ovaries massaged. Swabbed vagina with Lugol's sol.	1	1-17-29	9-23-29 249 da.	59 lbs. Appeared healthy	Pregnant again to first service
233 11-18-25	+	2-4-28 280 da. (1)	Calf (N)		Retained c. l.	6-12-28	C. l. expressed from each ovary	1	9-11-28	6-21-29 283 da.	Normal	
167 12-22-22	+	2-12-26 288 da. (1)	Calf (N)	3	Functional disturbances of ovaries	10-24-27 11-1-27 3-21-28	Douched uterus with 2% Lugol's sol. Dislodged c. l. Injected 5 cc follicular fluid subcutaneously Uterus massaged					Following 3-21-28 was turned out on pasture with a bull Slaughtered 10-29 and found pregnant

198 6-27-24 +	2-3-28 274 da. (2)	Calf (N)		Retained after-birth	2-4-28	Injected mineral oil and iodoform					
					2-6-28	Injected mineral oil					
					2-9-28	Injected 1000 cc 0.7% pepsin sol.					
				Catarrhal metritis	3-19-28	Uterus massaged and douched with 2% Lugol's sol.	1	5-26-28	1-23-29 242 da.	Died 4 hrs. after del.	*B. abortus* culture from calf
83 12-28-16 —	11-26-28 288 da. (1)	Calf (N)		Retained c. l.	1-15-29	C. l. expressed from each ovary	5				Slaughtered 9-18-29 Genital organs appeared normal on gross examination
89 4-10-19 +	12-14-25 280 da. (4)	Calf (N)	11	Lack of ovarian function	7-28-27	Uterus douched with 4% Lugol's sol.	3				
				Metritis cervicitis	10-11-27	Cervix swabbed with Lugol's sol.	1				Turned into pasture with a bull
				Lack of ovarian function	11-18-27	Injected 5 cc follicular fluid	2				Slaughtered 11-29 Not pregnant
				Granular vaginitis	2-4-28	C. l. expressed and vaginitis treated with Lugol's sol.					*B. abortus* recovered from both uterine horns
99 4-14-22 +	4-21-28 277 da. (5)	Calf (N)	4	Retained after-birth	4-23-28	Injected mineral oil and iodoform					
					4-26-28	Removed after-birth					
				Chronic catarrhal metritis	1-23-29	Douched uterus and vagina with 1000 cc 0.85% salt sol.	2				Slaughtered 9-18-29
191 5-7-24 +	2-12-29 280 da. (3)	Calf (N)		Pyometra	3-14-29	Uterus massaged. C. l. of last pregnancy expressed from left ovary					
					3-19-29	Uterus massaged					
				Lack of ovarian function	3-28-29	C. l. expressed	2				Slaughtered 7-28-29

TABLE I—*Continued.*

Previous to Treatment					Period of Treatment			Subsequent to Treatment				
Cow and Date of Birth	Reaction to Agg. Test for Br. Abortus	Termination, Length of Gestation and Parturition Number*	Sequelae	Services Following Last Parturition	Diagnosis of Condition	Date	Treatment	Services	Conception (Date)	Termination and Length of Gestation	Condition of Calf	Remarks
196 6-3-24	—	6-13-27 284 da. (1)	Calf (K) Stiff joints	3	Functional disturbances of ovaries	2-23-28	C. l. expressed from left ovary					
						3-16-28	C. l. and cyst expressed from right ovary					
					Slight catarrhal cervicitis	3-22-28	Douched cervix and vagina with 0.85% salt sol.	3	10-31-28	8-29-29 302 da.	Normal	
204 8-14-24	+	3-12-27 178 da. (1)	Calf (D)	3	Pyometra	8-20-27	Douched uterus with 2% Lugol's sol.	1	9-9-27	6-27-28 293 da.	Normal	Calved normally 3rd par. to one service
223 7-29-25	+	8-28-28 243 da. (1)	Calf (D)		Retained after-birth	8-30-28	Injected mineral oil and iodoform	5	3-24-29			Slaughtered 1 month after conception
						9-1-28	Removed after-birth					
228 8-17-24	?	1-15-29 289 da. (2)	Calf (N)		Slight pyometra	2-25-28	Expressed c. l. from left ovary					
						3-19-29	Douched uterus with 2% Lugol's sol.	2	6-2-29			Pregnant

231 10-1-25	+			9	Functional disturbances of ovaries	1-20-28	Injected 10 cc follicular fluid	1	1-25-28	10-16-28 264 da.	Dead at del.	
					Pyometra	12-18-28	Douched uterus with 2% Lugol's sol.					
					Pyometra	1-3-29	Douched uterus with 0.85% salt sol. and douched with 4% Lugol's sol.					
					Pyometra	1-11-29	Douched with 4% Lugol's sol.					
					Pyometra	3-15-29	Douched uterus with 0.85% salt sol. and injected colloidal iodin suspended in oil					
					Pyometra	3-28-29	Treated as above using an equivalent of 2 gms. of free iodin					Slaughtered 6-19-29
214 2-24-25	—	2-10-28 280 da. (1)	Calf (N)		Retained after-birth	2-13-28	Injected 1000 cc 0.7% pepsin sol.					
						2-21-28	Removed after-birth	1	6-16-28	3-18-29 275 da.	Normal	
					Retained c. l.	6-12-28	Expressed c. l. from right ovary					
189 10-28-23	+	9-10-27 288 da. (2)	Calf (N)		Slight catarrhal cervicitis	4-4-28	Douched uterus with 0.85% salt sol.	2	4-26-28	2-9-29 289 da.	Normal	
		2-9-29 289 da. (3)	Calf (N)		Retained after-birth	2-12-29	Injected mineral oil and iodoform					Slaughtered 11-12-29 Appeared normal on postmortem examination
						2-14-29	Removed after-birth	2				
G1 11-13-25	—	11-27-28 270 da. (2)	Calf (N)		Retained after-birth	11-29-28	Injected mineral oil and iodoform	3	4-18-29	1-21-30 278 da.	Normal	
						12-4-29	Removed after-birth					
G3 1-10-26	—	12-27-28 265 da. (2)	Calf (N)		Pyometra	1-11-29	Douched uterus with 0.85% salt sol.	4	5-22-29			Pregnant'
F1 9-16-25	—	8-20-27 269 da. (1)	Calf (N)		Retained after-birth	8-23-27	Injected mineral oil and iodoform	1	12-21-29	9-6-28 258 da.	Appeared healthy	Calved normally 3rd par. to one service
						8-26-27	Injected mineral oil and iodoform					

TABLE I—*Continued.*

Previous to Treatment					Period of Treatment			Subsequent to Treatment				
Cow and Date of Birth	Reaction to Agg. Test for Br. Abortus	Termination, Length of Gestation and Parturition Number*	Sequelae	Services Following Last Parturition	Diagnosis of Condition	Date	Treatment	Services	Conception (Date)	Termination and Length of Gestation	Condition of Calf	Remarks
O6 6-6-26	—	5-22-28 251 da. (1)	Calf (S, W and B)		Retained c. l.	7-10-28	Expressed c. l. from left ovary	1	7-13-28	4-17-29 278 da.	Normal	
O8 12-20-26	—				Lack of ovarian function	6-8-28	Injected 10 cc of follicular fluid	3	9-4-28	6-3-29 272 da.	Normal	
M218 9-15-22	—	6-17-28 273 da. (4)	Calf (N) Retained after-birth	3	Productive cervicitis	3-14-29	Trimmed external fold of cervix	1	4-1-29	12-30-29 274 da.	Normal	
M332 2-24-26	—	2-10-28 266 da. (1)	Calf (N)		Pyometra	4-11-28	Douched uterus with 2% Lugol's sol.					
					Pyometra	4-18-28	Treated as above					
					Pyometra	4-20-28	Pus drained, douched uterus with 2% Lugol's sol. and iodoform, bismuth sub-nitrate and mineral oil injected					
					Pyometra	5-4-28	Treated as above					
					Pyometra	6-5-28	Douched with 0.2% Lugol's sol.	3				
					Pyometra	11-1-28	Douched with 0.1½% Lugol's sol	2	1-5-29	9-30-29 268 da.	Normal	Slaughtered

M333 2-25-26	—	2-4-29 270 da. (2)	Calf (N)		Retained after-birth	2-5-29 2-7-29	Injected mineral oil and iodoform Removed after-birth					Died 6-12-29 Toxemia from chronic infections
41 5-10-19	—	6-11-27 264 da. (5)	Calf (D)	1	Catarrhal metritis	11-21-27	Douched uterus with 0.2% Lugol's sol.	2	12-8-27	9-1-28 267 da.	Dead	Slaughtered
130 4-28-20	—	9-12-27 286 da. (5)	Calf (N)		Retained after-birth Productive cervicitis Abscess on wall of uterus	9-14-27 9-26-27 12-19-27	Injected mineral oil and iodoform Injected mineral oil and iodoform Cervical canal filled with powdered boracic acid several different times	 3	 2-9-28			 Slaughtered 2 months following conception
166 12-19-22	+	6-18-27 216 da. (2)	Calf (D)	5	Catarrhal metritis	2-1-29	Douched uterus with 0.85% salt sol.	2				Slaughtered 4-17-29
95 3-12-20	+	8-1-27 112 da. (6)	Calf (D)	3	Atrophic endometritis Antrophic endometritis Slight hypertrophy of vaginal cervix	2-23-28 3-20-28 7-10-28	Expressed c. l. from left ovary Massaged uterus Douched uterus with 0.85% salt sol.	 1 2	 8-24-28			 Slaughtered 1 month following conception
208 10-14-24	+	8-11-27 217 da. (2)	Calf (D)		Retained after-birth	8-13-28 8-16-28 8-19-28	Injected mineral oil and iodoform Injected mineral oil and iodoform Injected mineral oil and iodoform					 Slaughtered Periuterine abscess

TABLE I—*Concluded.*

Previous to Treatment					Period of Treatment			Subsequent to Treatment				
Cow and Date of Birth	Reaction to Agg. Test for Br. Abortus	Termination, Length of Gestation and Parturition Number*	Sequelae	Services Following Last Parturition	Diagnosis of Condition	Date	Treatment	Services	Conception (Date)	Termination and Length of Gestation	Condition of Calf	Remarks
210 12-6-24	—	12-6-26 283 da. (1)	Calf (N)	9	Functional disturbances of ovaries	1-20-28	Injected 10 cc follicular fluid	1				Slaughtered
211 12-9-24	—			10	Granular vaginitis Metritis	7-27-28	Massaged uterus and ovaries Vaginitis treated with Lugol's sol. several times	3				Slaughtered *B. abortus* isolated from udder
222 2-28-25	+	12-18-27 253 da. (1)	Calf (D)			7-5-28	Douched uterus and vagina with 0.85% salt sol.	1				Slaughtered 10-15-28
M242 5-17-23	—	5-25-28 247 da. (3)	Calf Appeared healthy		Retained c. l.	2-13-29	Expressed c. l. from right ovary	1	2-17-29			Slaughtered 1 month following conception
M252 10-15-23	—	2-17-28 271 da. (3)	Calf (N)		Pyometra	3-19-28	Douched uterus with 0.2% Lugol's sol.	2	6-20-28			Slaughtered 6 months following conception

*The number of the parturition is given in parenthesis. Example: 5 = fifth parturition.
+ = positive. N = normal. W = weak.
— = negative. D = dead. B = blind.
? = suspicious. K = killed. C. l. = corpus luteum
S = small.

THE INCIDENCE OF GALL-STONES IN CATTLE*

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Bureau of Animal Industry, U. S. Department of Agriculture

Cholelithiasis is a general problem in biology, rather than a strictly human affair, just as are tuberculosis, cancer, arthritis, and numerous other pathologic entities; yet scant attention has been paid to the existence of gall-stones in animals. A recognition of the occurrence of gall-stones in animals will give to the subject a broader biologic outlook and will undoubtedly assist in the interpretation of the problem.

In animals, Feldman[1] cites Totten as reporting the occurrence of gall-stones in 23 cattle, out of a series of 5725, which were slaughtered in a Minnesota packing-house. The percentage incidence of gall-stones was 0.4 per cent. Totten observed that the stones occurred mostly in older animals which were generally in poor condition. Meyer, Nielson and Feusier[2] found that rabbits injected with typhoid bacilli sometimes developed gall-stones.

The writers undertook the study of the incidence of gall-stones in cattle intended for human consumption coming to a packing-house in Denver. Due to the facilities under which the study was conducted, observations were made on certain days when it was convenient for the study. On these days every animal of the species passing through the packing-house was studied. In this manner the incidence of gall-stones in a series of 2067 unselected animals was observed.

It is seen from the tables that 2067 unselected cattle were examined and that gall-stones were found in 21 animals (1 per cent). This is more than twice the percentage given by Feldman[1] who reports an incidence of 0.4 per cent in the series by Totten. Further, out of the 21 cases, 17 occurred in adult cows that had calved, 8 of which were pregnant at the time. Although heifers, which are adolescent females, comprise the largest number in the series, no gall-stones occurred among them. Totten observed that gall-stones generally occurred in older animals which were in

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TABLE I—*Incidence of gall-stones in cattle arriving in a Denver packing-house.*

DATE (1931)	COWS	HEIFERS	STEERS	BULLS	TOTAL	ANIMALS HAVING GALL-STONES
3-10	67	36	1	2	106	1
3-11	75	0	10	1	86	2
3-12	34	0	17	11	62	0
3-13	14	50	0	1	65	0
3-16	30	52	0	5	87	1
3-17	22	6	21	4	53	2
3-18	0	77	0	3	80	0
3-19	18	31	0	0	49	1
3-20	41	31	19	9	100	1
3-21	58	44	42	1	145	0
3-23	0	73	0	0	73	0
3-25	16	34	13	10	73	2
3-26	27	62	6	7	102	0
3-27	32	17	14	0	63	1
3-28	27	21	0	0	48	2
6- 4	14	15	10	0	39	0
6- 9	45	13	8	1	67	0
6-10	17	30	0	0	47	2
6-12	33	16	30	3	82	1
6-18	3	34	0	1	38	0
6-19	37	37	0	3	77	0
6-23	25	29	1	1	56	1
6-30	30	30	20	3	83	2
7- 1	0	25	0	3	28	0
7- 2	20	37	20	3	80	0
7- 7	4	14	27	5	50	1
7-11	8	55	56	2	121	1
7-14	56	48	1	2	107	0
Totals	753	917	316	81	2067	21

Cows = female cattle which have already calved.
Heifers = female cattle which have not calved and which are between the ages of one and three years.
Steers = adult male cattle which were castrated before reaching the service age.
Bulls = adult male cattle which have reached the approximate age of two years.

poor condition. However, in our series, the majority of the animals were in fairly good condition, were well nourished and suitable for use as beef, and further, they were not advanced in years. The ages of the animals varied from 2 to 10 years, with an average age of 6 years, which cannot be considered an advanced age for cattle.

TABLE II—*Incidence of gall-stones in the different classes of cattle.*

	COWS	HEIFERS	STEERS	BULLS	TOTAL
Animals examined	753	917	316	81	2067
Animals having gall-stones	17	0	3	1	21
Percentage	2.3	0.0	1.0	1.2	1.0

TABLE III—*Pathology of gall-stones in cattle.*

CASE	AGE (YEARS)	MONTH OF PREGNANCY	STONES	GALL-BLADDER	ASSOCIATE PATHOLOGY
C1	4		1 large, several small	Several small papillomata in mucosa	
C2	7	3	3 large, several small	No gross pathology	Fluke infestation of liver; stones in duct
C3	7		1 large	No gross pathology	
C4	5	5	2 large	No gross pathology	
C5	5	3	1 large	No gross pathology	
C6	5	3	1 large	Reddish placques in serosa	
C7	10		3 large	Two diverticula	Epithelioma of eye; metastases to lungs
C8	7		9 large	No gross pathology	
C9	4	4	3	No gross pathology	
C10	10		4 large	No gross pathology	Stones in ducts
C11	6	5	1 large	No gross pathology	
C12	8	4	37 large	Several small papillomata in mucosa	
C13	8	3	1 large	Several small papillomata in mucosa	
C14	7		1 large	Several small papillomata in mucosa	
C15	7		1 large	No gross pathology	Abscess in liver
C16	7		2 large	No gross pathology	Stones in ducts
C17	10		10 small	Wall thickened, inflamed and indurated	Liver abscess adjacent to neck of gall-bladder
St1	3		2 small	No gross pathology	Stones in ducts
St2	4		5 small	No gross pathology	Stones in ducts
St3	2		1 small	Several small papillomata in mucosa	
B1			1 small	No gross pathology	

C = cow. St. = steer. B = bull.

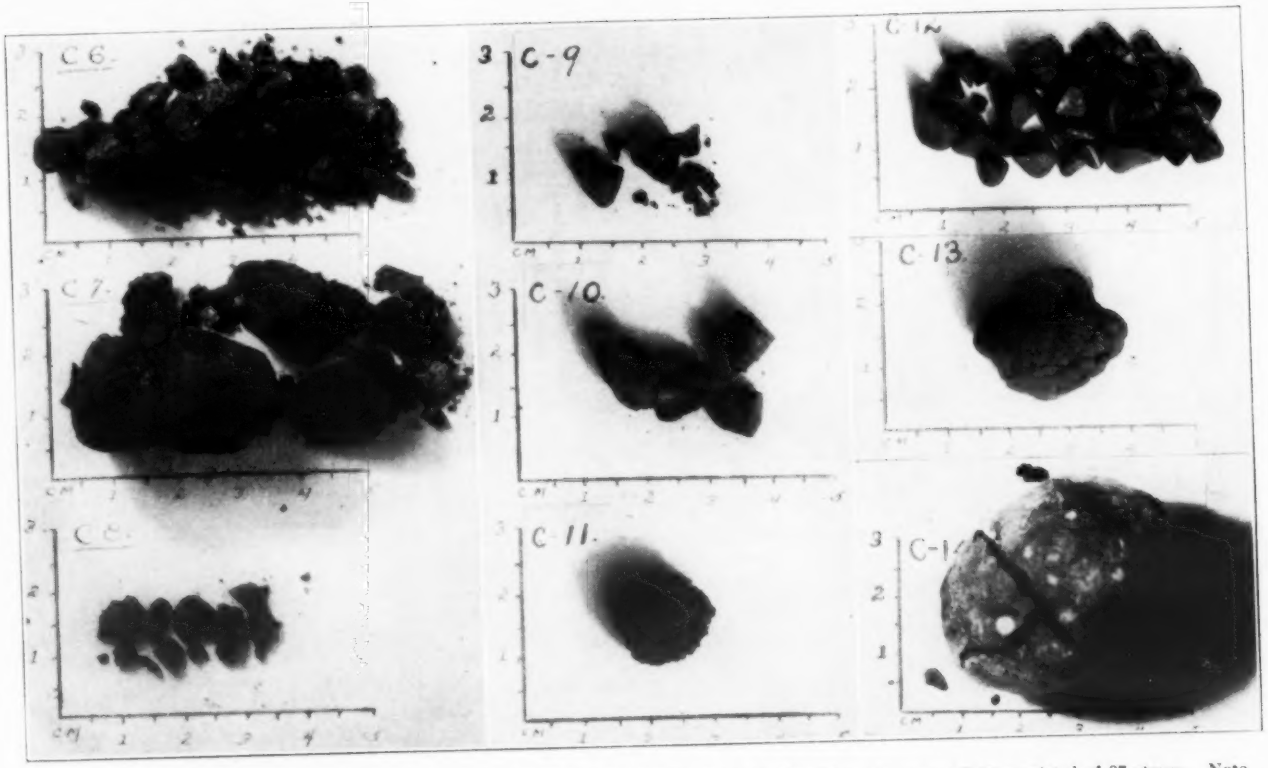

FIG. 1. Types of gall-stones found in cattle. Specimen C-14 was the largest stone in the series. Specimen C-12 consisted of 37 stones. Note variety of shapes in the different specimens. (Photographs are by courtesy of Dr. H. J. Corper, of Denver.)

The gall-stones varied in size from that of gravel to a small egg. The largest stone in this series measured 4.5 x 3.2 x 2.7 cm. The number of stones varied 1 to 30 or more. One gall-bladder contained 37 moderately large stones. In shape, the stones were variable. They were irregular, round, oval, mulberry or faceted in contour. In color they were all some shade of brown, either yellowish brown, greenish brown, chocolate brown or reddish brown. Generally they were extremely brittle and broke easily upon ordinary handling, revealing different types of structures. Some were stratified, some were of uniform and some were of mixed structure. They were much more friable than human gall-stones. Some of the stones floated when dropped in water, indicating a probable high cholesterol content, and some were slightly fatty to the touch. The specific gravity was not measured in every case, but they all felt relatively light for their mass.

The following gross pathologic changes were noted in the gall-bladders: five specimens contained small papillomatous nodules in the mucosa; 1 contained several small, reddish placques in the serosa, probably fluke incrustations; 1 gall-bladder contained 2 small diverticula; 1 was thickened and markedly indurated and adjacent to a liver abscess, and 13 showed no gross pathologic alterations.

In 17 of the specimens, the stones were found in the gall-bladders, while in 4 the stones were present in the bile-ducts.

Among the significant associate pathologic changes in the animals, the following were observed: One animal indicated fluke infestation of the liver; 1 had an epithelioma of the eye, with metastases to the lungs, while 2 had liver abscesses.

Summary

1. Cholelithiasis is a general problem in biology.

2. Scant attention has been paid to the incidence of gall-stones in animals.

3. A series of 2067 unselected cattle coming to a packing-house in Denver have been studied for the presence of gall-stones.

4. Gall-stones were found in 21 animals, about 1 per cent of the series.

5. Stones occurred most frequently in adult cattle that had been pregnant (cows).

6. Stones were found both in the gall-bladder and in the bile-ducts.

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STUDIES OF THE LIVER FLUKE (FASCIOLA HEPATICA)*

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INTRODUCTION

Since an intensive battle is being waged against the liver fluke in the western states, it is thought that it might be well to publish data having a direct bearing on the progress of this fight. Much information has been given to the world about the common liver fluke, but when one begins efforts to control this parasite, one immediately feels the need of new facts. It is the purpose of this paper to give such information as has been gained at this station since the publication of Oregon Station Bulletin No. 296, "Fascioliasis in Oregon Sheep and Goats," in June, 1930.

LENGTH OF TIME INFESTED SNAILS DISCHARGE CERCARIAE

Observations on the development of the parasites in the snail host prove that the time of development varies, according to temperature and perhaps other factors, from seven to fourteen weeks. Most of our experiments have indicated the time as nearer fourteen weeks than seven weeks.

In some cases cercariae were seen to swarm from snails over a very short period of discharge, but in most of the artificially infested snails the discharge of cercariae occurred over a considerable period. In one case, artificially infected snails discharged parasites for over two months. This batch of snails, as originally collected, was known to be free from infestation on the negative findings obtained by dissection and observation of representative specimens. The snails were then infected on November 22, 1930, with a limited number of miracidia. The miracidia were pipetted from the jar in which the eggs were hatching, care being used not to include partly developed eggs that might hatch at a later date. These snails, then about half-grown, began discharging cercariae on February 15, 1931, 83 days after exposure to miracidia, and continued to discharge them until April 20, 1931.

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Viability of Encysted Cercariae

Some of our advice to stockmen, in answer to their questions as to pasture infestations, has been based chiefly on opinions. Ross and McKay[1] quote Marek as showing that encysted cercariae were capable of living on moist hay for eight months. Evidence obtained at this station proves that encysted cercariae are capable of living under rather adverse conditions for eleven months.

On June 2, 1391, 150 encysted cercariae were fed to a mature rabbit. These cysts had been in the cold storage plant since July 9, 1930. The cysts were from naturally infested snails and were allowed to remain at their original points of encystment until the time of feeding. Some were certainly alive, because movement could be seen when the outer cyst-wall was removed. A good many were dead and this could be easily ascertained after the removal of the cyst-wall. On June 8, 1931, the rabbit was autopsied; no signs of fluke were found in the liver. Sinitsin reported that flukes remained in the peritoneal cavity for fourteen days; possibly the rabbit in our experiment was destroyed too soon. No attempt was made to determine whether parasites were present in the peritoneal cavity.

A second attempt was made to determine if these apparently alive cercariae would produce liver infestation. On June 9, 1931, 40 cysts were fed to each of three guinea pigs. Two of these guinea pigs were killed, June 15, 1931. Burrows were distinct in both livers. One fluke 500 μ long was recovered. The third animal was not destroyed.

The temperatures in the cold storage plant during the eleven months varied between —2.78° C. and 4.44° C., with an average temperature probably above freezing. The temperature went below freezing at least twice, according to our records. During the summer, for a two-month period, the temperatures were recorded with a maximum and minimum thermometer. The room was in total darkness except when lighted by electric lights, and was then lighted only for short periods of time. The cysts were kept moist by being covered with moist cotton. The dish containing the cysts was covered with a glass bowl to prevent evaporation.

The Effect of Copper Sulfate on Encysted Cercariae

Since the work of Chandler,[2] in 1920, and that of Walton and Jones,[3] in 1925, we have known that the snail hosts could be easily

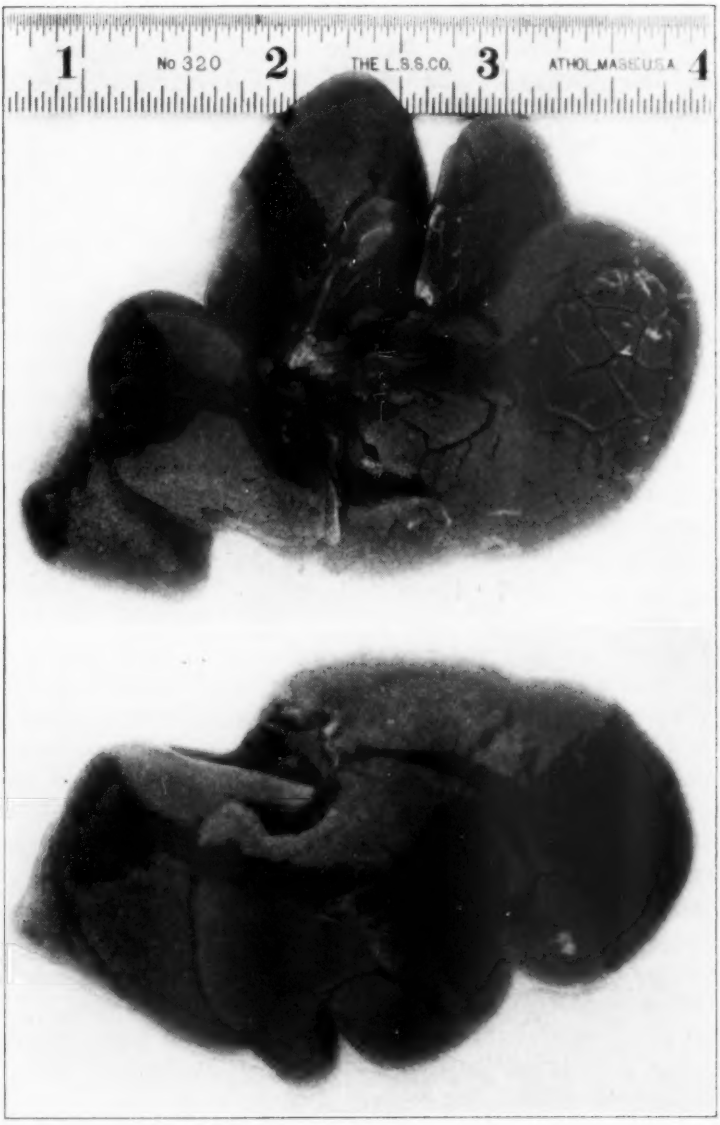

FIG. 1 (above). Liver from guinea pig fed cysts treated with a 1:2500 copper sulfate solution.
FIG. 2 (below). Liver from guinea pig fed cysts treated with a 1:500 copper sulfate solution.

destroyed by copper sulfate, even in very dilute solutions. No evidence has been available to show the possible effects of copper sulfate upon cercariae that might have encysted before treatment took place. In treating pastures with powdered copper sulfate, solutions made with surface water would vary considerably, hence in exposing encysted cercariae to solutions of copper sulfate, solutions varying from what was figured to be an average strength, up to a strength that would be poisonous if consumed in quantities such as a sheep would drink, were used. Guinea pigs were fed cysts exposed for 30 hours to solutions varying from 1:500 to 1:5000. Some of the cysts were removed from their points of encystment, while others were allowed to remain on plant life growing in aquaria.

Figure 1 shows the liver from the guinea pig fed cysts which had been treated with a 1:2500 solution of copper sulfate, while figure 2 shows the liver from the guinea pig fed cysts which had been treated with a 1:500 solution. Both livers show fewer burrows than the liver from the guinea pig fed untreated cysts, even considering the fact that the check animal was fed a greater number of cysts.

TABLE I—*Feeding experiment on guinea pigs.*

GUINEA PIG	CYSTS FED	COPPER SULFATE SOLUTION	RESULTS
1	20	1:5000 (organic material present)	+++
2	30	1:5000 (pure solution)	+++
3	40	1:2500 (organic material present)	++
4	40	1:1000 (organic material present)	++
5	40	1:500 (organic material present)	+
6	70	Water (organic material present)	++++

+, ++, +++, ++++ = burrows varying from one to very many.

On May 27, 1931, eight days after feeding, all guinea pigs were killed. Burrows or fluke marks were found in all livers. In g.p. 5 only one burrow could be found, while in g.p. 3 many burrows were present. The fact that some parasites had encysted on organic material did not make any noticeable difference. No special effort was made to recover flukes from these livers. Those recovered measured 900 μ when extended.

MIGRATION OF YOUNG FLUKE IN HOST

Sinitsin,[4] in 1914, and Shirai,[5] in 1927, showed that the course of the young parasite in its efforts to reach the liver is through

the peritoneal cavity. Sinitsin washed flukes from the peritoneal cavity of artificially infested animals at various times after feeding cysts. Measurements of the young flukes and the absence of parasites in the bile-ducts assured him of the true course of the young worm.

Shirai[5] succeeded in uniting the peritoneal cavities of two guinea pigs. Upon feeding cysts of the liver fluke to one of these pigs he was able to find burrows of the young worm in the liver of the other guinea pig. Data obtained at this station substantiate the findings of both these investigators.

On September 26, 1930, a number of cysts were fed to a mature guinea pig. These cysts were collected August 5, 1930, and September 3, 1930. On September 29, 1930, three days after feeding, 33 live flukes were washed from the abdomen of the infested guinea pig. One immature fluke had already entered the liver. Twelve of these flukes were injected into the peritoneal cavity of a second guinea pig immediately. On October 16, 1930, this second guinea pig was destroyed and one fluke, 3 mm. long, was recovered from its liver.

On March 2, 1931, 361 cysts, collected from artificially infested snails, were fed to lamb 33. On March 5, 1931, lamb 33 was destroyed and 43 young flukes were washed from the peritoneal cavity. No parasites had entered the liver. Subserous hemorrhages and tags (shreds of connective tissue) on the intestine were found. Fifteen of the flukes washed from the peritoneal sac of lamb 33 were injected into the peritoneal sac of lamb 6 on the same date. On March 21, lamb 6 was destroyed and 3 flukes, averaging 2 mm. in length, were recovered from the liver. The scars and burrows would indicate that more parasites had reached the liver than were recovered. This lamb was corral-raised and fed on hay obtained from fluke-free pastures.

Attempts to secure infestation of the liver by injection of live flukes into the jugular vein of a lamb and into the thoracic cavity of a goat gave negative results. Hemorrhagic areas were found in the lungs in both, but no flukes were recovered. Sections of these areas also failed to reveal parasites.

In one experiment, 246 flukes were washed from the peritoneal cavity of a guinea pig. These flukes were recovered from the animal three days after feeding 900 cysts; none had penetrated the liver.

Where a count of cysts was made at the time of feeding, between 8 and 30 per cent of the parasites fed were recovered. Young

flukes recovered from the peritoneal cavity of experiment animals had considerable vitality and remained alive in a saline solution at room temperature for 24 hours.

Discussion

Much work has been done on the development of the parasite in the intermediate host. Most of this work has indicated this development to be influenced by surrounding conditions. It was surprising to see snails discharge cercariae for more than two months from the time the first cercariae were noticed swimming in the aquarium. Such evidence indicates the possibility of cercariae existing in infested snails through winter months or months of drought and still being capable of being discharged when favorable conditions permitted.

The evidence as to the viability of encysted cercariae substantiates the findings of Marek, as noted by Ross and McKay, and adds to our knowledge of what we might expect from pasture infestations. While the information obtained was not gained under pasture conditions, one can readily conceive of conditions similar to ours existing on parts of some pastures.

Knowing that copper sulfate will destroy encysted cercariae lends further argument in support of the present fluke-control measures. No doubt only a small part of the solutions formed by the present use of copper sulfate would be of 1:500 strength, but these solutions might destroy large numbers of cercariae. One can readily conceive of a 1:500 solution filling a cow-track containing 18 to 20 snails, the majority of which had each discharged several hundred cercariae, which had encysted on the surface of the water and on the plant life growing there. No doubt the solutions made by our present methods of using copper sulfate are highly variable.

In dealing with the above minor points, it is thought that the major parts of the fluke-control problem become more approachable. Different theories as to the migration of the parasite in the final host have received equal support from equally important investigators, although the theories of May, Spinola, Gerlach, Sinitsin, and Shirai had the greatest following. Knowing the exact method by which the parasite reaches the liver will help explain some of the pathology we find in infested animals.

Summary

1. Cercariae from artificially infested snails were discharged four months and twenty-seven days after infestation.

2. Cercariae were shown to remain alive and infective for eleven months after their discharge from snails.

3. Copper sulfate in solutions of 1:500 destroyed encysted cercariae.

4. Our experiments confirm previous findings to the effect that the route of migration of young flukes in the final host is by way of the peritoneal cavity from the intestine to the liver.

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New Helminthological Periodical

A new quarterly, *Helminthological Abstracts*, the first number of which appeared in April, 1932, has been issued as a supplement to the quarterly *Journal of Helminthology*. It contains a résumé of current periodical literature in its field, and is published by arrangement with the Imperial Bureau of Agricultural Parasitology, at St. Albans, England. Both periodicals are edited by Dr. R. T. Leiper, director of the Bureau.

The new publication will make possible the early reviewing of current helminthological literature, and will contain, in addition to between 80 and 100 abstracts in each issue, the Notes and Memoranda which have heretofore appeared in the *Journal*. Subscriptions to *Helminthological Abstracts* are 16s. 6d., but orders from subscribers to the *Journal* will be accepted at 15s. a year, post free. Address the *Journal* at the London School of Hygiene & Tropical Medicine, Keppel Street (Gower Street), London, W. C. 1, England.

Veterinarians to Visit Ozarks

A trip to the Ozark Mountains, noted "Shepherd of the Hills" country, has been planned to follow the annual meeting of the Missouri Veterinary Medical Association this year. The meeting will be at Springfield, July 20-21, and the local committee has arranged the Ozark outing for July 22. Details may be obtained from Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

LESIONS IN THE STOMACH OF A DOG SIMULATING ACTINOMYCOSIS*

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Lesions designated as actinomycosis occasionally are encountered in dogs. After reviewing published reports on the subject, one obtains the impression that the etiologic agent in such cases is variable and unlike that usually attributed as the cause in the bovine. Actinomycosis in dogs is commonly referred to as streptothricosis. It is more or less chronic, usually traumatic in inception, and is characterized as a purulent, hemorrhagic, phlegmonous condition with formation of fistula. The tissues overlying the thorax are usually affected, and frequently the thoracic cavity and lungs are involved. Marked pleuritis is present and the parietal pleura is often covered with soft, pendulous, villous-like proliferations. A cloudy, yellowish-red fluid, containing many sand-like granules, grayish white to yellow, is usually present in the thoracic cavity and occasionally in the abdomen. Associated with the lesions there may be long, slender, Gram-positive, branching filaments that show a tendency to form club-shaped terminals; these organisms are considered of etiologic significance. The formation in the tissues of ray fungi or actiniform colonies, which are characteristic of actinomycosis in the bovine, does not occur. Several cases of this kind have been described in detail by Bahr, Kitt and others, since the disease was first reported by Rivolta and by Rabe. The disease is also described briefly by Nieberle and by Hutyra and Marek.

The material which is the basis of this report was unusual in that the etiologic agent was represented by structures that in many respects resembled the ray fungus observed in pus and tissues from lesions of actinomycosis in cattle. The lesions were limited to the wall of the stomach, which was also unusual, since the stomach is seldom involved in chronic infectious granulomatous lesions.

HISTORY OF CASE

The animal was an adult mongrel female dog, weighing, when first observed, 12.2 kg. In August, 1927, the dog was placed under complete, general anesthesia and the abdomen was opened with the intention of making an Eck fistula. The operator, finding that the stomach was

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strikingly abnormal, abandoned the original objective and explored the abdomen instead. The stomach measured approximately 30 cm. from the esophagus to the pylorus, and from the greater curvature to the lesser curvature, about 20 cm. It was firm and leathery and on palpation several areas of induration were encountered which seemed to be confined to the wall of the stomach and did not extend beyond the mucosa. The pylorus appeared normal and there was no evidence of stenosis. The regional lymph-nodes of the stomach were enlarged and firm; significant abnormalities of other abdominal organs were not observed. Biopsy was not done and the condition was considered grossly to represent linitis plastica. The incision was closed and the animal was kept for further observation, particularly for the development of metastasis.

Two years later, the animal appeared to be in excellent condition and weighed 14.2 kg. It was again anesthetized and the stomach was explored; the condition was practically the same as it had been two years previously. The wound was repaired and the animal recovered uneventfully. About a year later, the animal was used for a physiologic experiment involving the thyroid gland and was noted to be in good physical condition. Five months later, or approximately three years and seven months after the lesions of the stomach were first observed, the dog was killed.

Necropsy disclosed a few adhesions of the liver, spleen, and intestine and over a portion of the stomach. In the submucosa of the greater curvature was a large, round, somewhat flattened mass of rather firm consistence. It measured 17 by 14 by approximately 4 cm. Superficially the mass appeared to be a single unit but closer examination showed it to consist of one large and two smaller units that were more or less intimately united. The regional lymph-nodes did not appear enlarged.

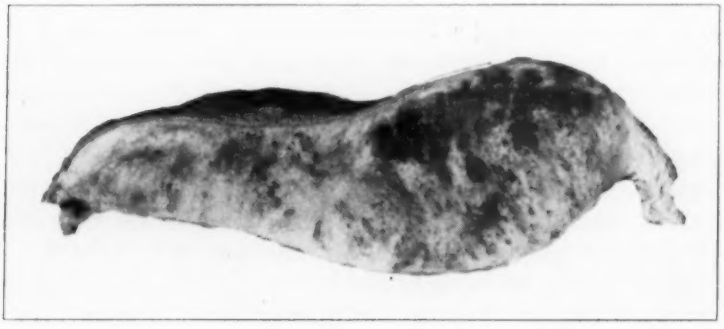

FIG. 1. Cross section of the wall of the stomach of a dog. The granulomatous thickening, which has practically obliterated all the normal structures of the organ, is shown. The intact mucosa is plainly discernible.

GROSS PATHOLOGIC OBSERVATIONS

In the mucosa overlying the indurated mass were several small, superficial, irregular ulcerated areas, but otherwise the mucosa was intact and without gross evidence of disease. A transverse incision through the larger indurated mass revealed the extremely fibrous character of the structure. The lesion included the entire width of the wall of the stomach; the line of contact of the new tissue with the mucous layer was very definite (fig. 1). In some portions of the lesion, just beneath the mucosa,

there were several cavitations of irregular contour which contained a sticky, grayish-white, purulent material. Similar material, but less in amount, exuded from much of the surface of the freshly cut lesion. An unstained, crushed preparation of a portion of the purulent material obtained from the lesion and examined in the fresh state revealed enormous numbers of actiniform structures not unlike those in pus from lesions of actinomycosis in cattle.

Animal Inoculations and Cultures

Emulsions were prepared of portions of the purulent material containing the ray fungi and these were injected subcutaneously, intraperitoneally, and in a few cases intracerebrally into dogs, guinea pigs and rabbits. Although some of the animals were permitted to live for six months subsequent to inoculation, in none were significant lesions produced.

Attempts to isolate the etiologic agent by cultural means also failed. Inexperience in methods most favorable for promoting the growth of Actinomyces may have been in part responsible for our failure to secure a culture of the organism, although considerable attention was given to this phase of the study.

Pathologic Histology

Sections were prepared for histologic study from practically all organs besides the involved stomach. There was no sign of the disease in any of the tissues from the other parts of the body; the infection apparently was limited to the wall of the stomach at the greater curvature. Although the gastric mucosa was intact in all the sections of the stomach that were examined, the normal contour of the nonmucous part of the organ was greatly altered in those portions which were involved by the disease. The muscular coat was not discernible in many fields and in others only remnants of it remained. Practically the entire muscular portion of the wall of the stomach occupied by the lesions had been replaced by a proliferation of granulomatous fibrous connective tissue. The serosa remained. Although much of the fibrous connective tissue was laid down in dense strands that were disposed in many different directions and possessed a generous vascular system, many purulent areas were present in which the connective tissue was minimal or absent. The abscesses were not circumscribed and irregular spaces or sinuses filled with a purulent cellular content were scattered promiscuously throughout. The cellular exudate consisted principally of eosinophiles,

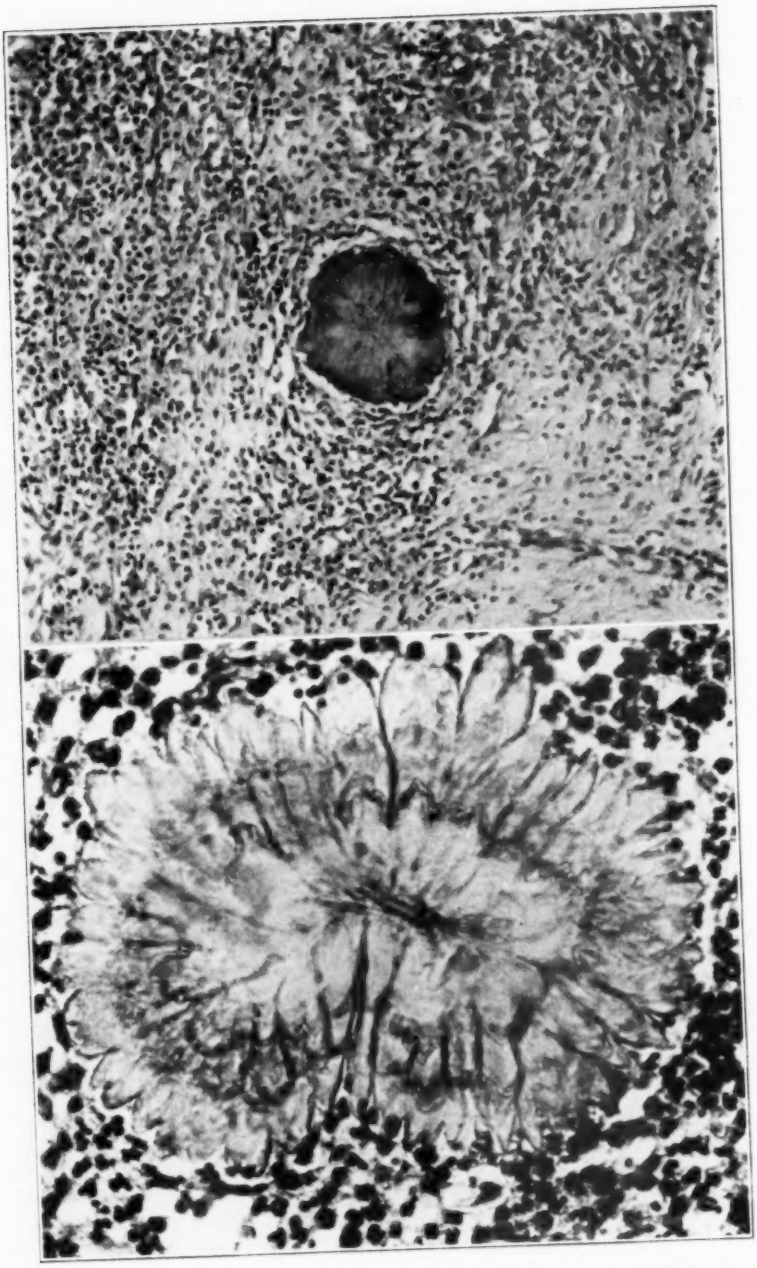

FIG. 2 (above). Actiniform granule in the midst of granulation tissue. Many leukocytes are present (x 130).

FIG. 3 (below). Radiating colony surrounded by many leukocytes (x 525).

macrophages and monocytes. Few polymorphonuclear leukocytes occurred and foreign-body giant-cells were not observed; there were few lymphocytes.

Distributed irregularly throughout much of the exudative portions of the tissue were numerous actiniform structures (figs. 2 and 3). Although these forms varied somewhat in structural detail, they possessed many significant features in common. They consisted for the most part of forms simulating ray fungi, and were composed of rather coarse club-like structures arranged in a radiating fashion (fig. 4*a*). They stained decidedly acidophilic and were ovoid to spherical. Their size was somewhat variable, as is indicated in table I, showing the measurements of ten of these forms.

TABLE I—*Sizes of club-like structures.*

GREATER DIAMETER (MICRONS)	LESSER DIAMETER (MICRONS)
102	64
101	141
158	144
68	50
152	147
117	114
132	110
114	88
117	102
94	70

Although the respective component parts of the majority of the radiating structures tapered to a more or less blunt point at the terminals, a few actiniform masses were found in which the respective constituents were bulbous. The latter gave the structure, of which it was a part, a rosette appearance (fig. 4*b*). At the periphery of many of the radiating structures there was evidence of a definite attempt on the part of certain monocytic cells to establish a cellular investment over all or a part of the actiniform mass. As a result of the uniting of the monocytes a slender cellular membrane surrounded some of the forms, and in other instances a thick multinuclear protoplasmic mass occurred over a portion of the periphery; these were the nearest approach to foreign-body giant-cells observed (fig. 4*b*).

COMMENT

From the information obtained from this study it is impossible to determine with certainty the specific nature of the etiologic

factor responsible for the lesions. The numerous actiniform granules in the unfixed pus and in the histologic sections of the lesions seem of etiologic significance, yet the failure to induce comparable lesions in experiment animals and to isolate by

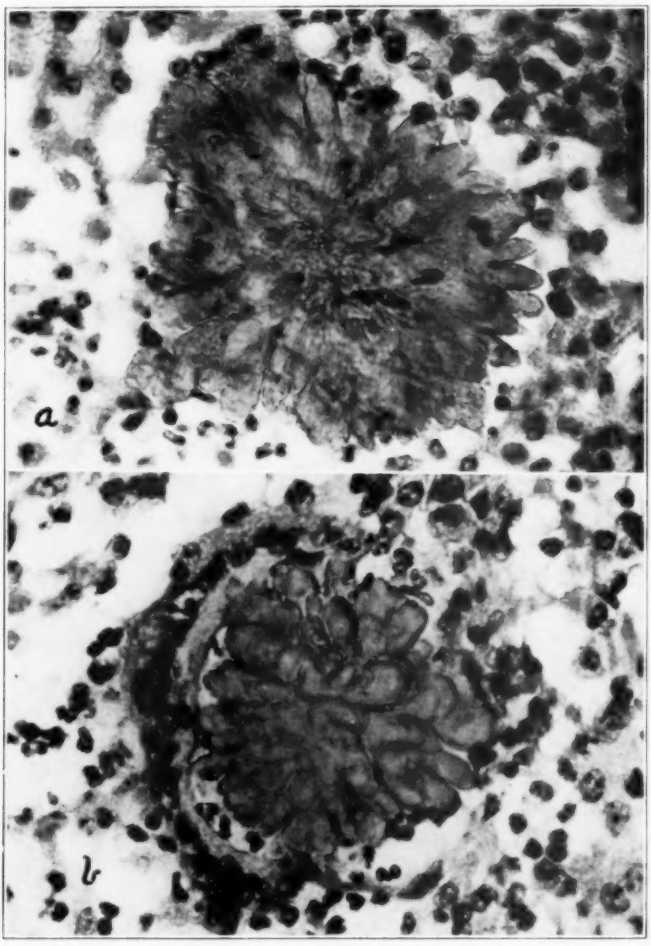

FIG. 4. *a* (above), cluster of club-like forms simulating the ray fungus of actinomycosis; *b* (below), actiniform granule consisting of bulbous or club-like radiating structures. A multinuclear protoplasmic mass is present over a portion of the periphery (x 525).

cultural means a microörganism such as one might expect from material containing actiniform granules of this kind, leaves the exact identity of the causative microörganism undetermined.

The condition suggests in many ways an actinomycotic infection, but a study of the material revealed several points of dis-

similarity with the usual picture of this disease. If one assumes the ray fungus-like granules to be of etiologic significance, it is of course evident that the condition is not a streptothricosis such as occurs in the lesions of dogs affected with what may be known clinically as actinomycosis. Again, if the lesions in this instance represent a true actinomycotic infection, the morphology of the etiologic agent is at variance with the organism of actinomycosis as it occurs in the tissues of cattle and hogs spontaneously affected with this disease. The actiniform masses are larger and the respective units constituting the various actiniform structures are different from those observed in the granules of *Actinomyces bovis*. The radiating club-like forms are larger and coarser and the bulbous swellings observed in some of the rosette-like granules are unlike anything that occurs in the granules of actinomycosis, such as is found in lesions from the bovine or from swine.

The failure of the respective actiniform granules to become the center of a definitely circumscribed focal lesion such as characterizes true actinomycosis was an interesting feature of the histopathology. The cellular response also was different from that which occurs in lesions of spontaneous actinomycosis of cattle or swine. Epithelioid cells were few, and an excessive amount of eosinophiles with many monocytes and macrophages constituted a cellular reaction not usually seen in ordinary actinomycotic infection. The absence of giant-cells of the foreign-body type was also in marked contrast to lesions of actinomycosis, since these structures are frequently present in large numbers in this condition. It must be recognized, however, that the body's reaction to a given microörganism is not necessarily constant in all animals susceptible to that organism, and the character of the cellular response to a certain infective agent is not determined entirely by factors inherent in the infective agent but is subject to variations which are introduced by the mechanism controlling the body's cellular defense. With these possibilities in mind, one might expect certain minor dissimilarities in the lesions of actinomycosis in different species if the infective agent were identical in each instance. In this case, however, both the cellular response and what would seem to be the etiologic parasite are at variance with the usual pathologic changes one is accustomed to see in spontaneously infected cases of actinomycosis of cattle and swine.

Assuming the actiniform granules observed in the lesions of this case to be of etiologic significance in the genesis of the disease, there are at least two possibilities as to the true character of these structures. They may represent a pathogenic species of the genus Actinomyces different from *Actinomyces bovis* or *Actinomyces hominis*, or these radiating forms may have been produced by bacteria. Henrici pointed out that both the lesions and the characteristic granules of actinomycosis may result as a consequence of certain forms of bacteria; he also stated:

> The mere finding of radiating lobulated granules in pus is not sufficient evidence to establish a diagnosis of actinomycosis. One must further prove that these granules are composed of fine branched filaments characteristic of the mycelium of Actinomycetes.

Our failure to cultivate the organism or to demonstrate the presence of branching filaments makes a designation of actinomycosis difficult to substantiate on other than clinical and morphologic grounds. Such proof is suggestive and not convincing.

The chronicity of the disease and its failure to metastasize or to involve the regional lymph-nodes are indicative of infection of slight virulence. There was no evidence that the disease seriously interfered with the functional capacity of the involved stomach nor did the markedly enlarged organ appear to cause the animal discomfort or distress. Digestion was not suspended or seriously interfered with, since the animal continued in excellent physical condition and in fact gained weight while maintained on the routine kennel ration. This was probably due to the fact that the disease was limited to the wall of the stomach and did not violate the mucosa.

The occurrence of genuine actinomycotic lesions in the stomach is encountered but rarely even in the bovine, which is most commonly affected with this disease. A few instances have been reported, however, in which the abomasum of bovines was the primary site of the infection which probably had its inception as a consequence of an abrasion of the gastric mucosa. According to Joest, the disease may assume a nodular, tumor-like or a diffuse form. Schlegel found an actinomycotic tumor weighing 2 kg. in the true stomach of a four-year-old bovine, and he also observed, in another bovine, diffuse thickening of the walls of the abomasum and numerous small nodules in the submucosa which were due to an actinomycotic infection. Joest described thickening of the walls of the true stomach of a two and one-half year-old bovine as a consequence of an actinomycotic process. Such cases are unusual, which seems remarkable considering the

susceptibility of the tissues of cattle to *Actinomyces bovis* and the common occurrence of lesions of the disease in other tissues of these animals.

Summary and Conclusions

An extensive infective granulomatous condition limited to the wall of the greater curvature of the stomach of a dog was observed at intervals for about three and one-half years. The duration of the disease was uncertain, but it was characteristic of chronicity and of slight progressiveness. The infective character of the lesions was not ascertained until the animal was killed for necropsy.

The purulent exudate obtained from the lesions contained numerous radiating colonies similar in many respects to the so-called sulfur granules of actinomycosis, and histologic sections revealed many actiniform structures that were apparently of etiologic significance. Attempts at culture and animal inoculations were not successful in isolating or demonstrating a pathogenic microörganism from an emulsion prepared from the diseased tissues. Although the lesions were somewhat at variance with those usually observed in actinomycotic infections, the clinical course of the infection and the morphology of the actiniform masses were suggestive of this disease. The exact nature of the infective agent responsible for the pathologic changes remains in doubt, since it is recognized that radiate granules resembling those of true actinomycotic infections may also be formed by microscopic parasites other than Actinomycetes.

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Doctor Moskey Exposes Misbranded Nostrums

The recently issued report of the third annual meeting of the Maryland Stockmen's Association, held in Baltimore, January 6-7, 1932, reproduced in full the paper, "Misbranded Veterinary Preparations," presented at the meeting by Dr. H. E. Moskey, of the U. S. Food and Drug Administration.

THE DIFFERENTIATION OF PASTEURELLA AVICIDA AND BRUCELLA INFECTIONS IN THE FOWL*

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Recently Mallmann[1] has reported on the interagglutinability of members of the Brucella and Pasteurella genera. Huddleson and Emmel,[2,3] Emmel,[4] McNutt and Purwin[5,6] and Gilman and Brunett[7] have reported the finding of natural reactors to the agglutination test for the species of the genus Brucella among farm fowls. The latter authors and one of us (Emmel[4]) have reported the isolation of a species of the genus Brucella from naturally infected birds obtained from farm flocks. However, it is the opinion of Mallmann[1] that the fowl is an unnatural host for the species of the genus Brucella and that natural reactors to the agglutination test for Brucella infection may be the result of antibodies produced in birds by organisms which might cross-agglutinate Brucella antigens. Mallmann's report shows that there is cross-agglutination of the Brucella and Pasteurella genera. It is therefore the purpose of this paper to study these infections in the fowl in an attempt to differentiate the two types of infection.

CROSS-AGGLUTINATION STUDIES

Experiment I: A study was first made of *P. avicida* and Brucella antigens on the sera of birds injected with various strains of *P. avicida*. Seven strains of this organism were used in this study. Strain I was secured from the U. S. Department of Agriculture; II, from Dr. H. J. Stafseth, Michigan State College; III, Iowa State College; IV (fluorescent type) and V (blue type), from Dr. L. T. Webster, Rockefeller Institute; VI, American Type Culture Collection, and VII, from Dr. L. D. Bushnell, Kansas State College.

Twenty-three adult birds were divided into seven groups of three birds each and one group of two birds. (See table I.) One-half cubic centimeter of a saline suspension of *P. avicida* of a density equal to McFarland's nephelometer tube 1 was injected intravenously into each bird. A different strain of *P. avicida*

*A part of this work was done in the Laboratories of the Department of Bacteriology, Michigan State College. Published with the permission of the Director of the Alabama Agricultural Experiment Station. Received for publication, December 11, 1931.

was injected into each group of three birds. The two remaining fowls served as controls.

The blood serum from one bird (1071) failed to show the presence of agglutinins when tested with the seven *P. avicida* antigens. The blood sera of 29 birds reacted in a dilution of 1:25, 10 in a dilution of 1:50, 6 in a dilution of 1:100, 2 in a dilution of 1:200, 1 in a dilution of 1:800, 2 in a dilution of 1:1600, and 1 in a dilution of 1:3200. Ninety-six of the 147 tests failed to show agglutination. The results of the agglutination tests conducted on the same sera with a Brucella antigen* showed that the sera from 10 to 21 birds failed to react. Four samples reacted in a dilution of 1:25, 6 in a dilution of 1:50 and 1 in a

*Brucella antigen used in this laboratory in routine agglutination tests.

TABLE I—*Results of agglutination tests on sera of birds injected intravenously with various strains of P. avicida.*

BIRD	STRAIN OF PASTEURELLA INJECTED	HIGHEST TITRES SHOWING COMPLETE AGGLUTINATION*								P. AVICIDA ISOLATED AT AUTOPSY
		STANDARD BRUCELLA ANTIGEN	P. AVICIDA ANTIGENS							
			1	2	3	4	5	6	7	
1932	1	—	—	1:25	—	—	1:50	—	—	+
1887		—	1:25	—	—	—	—	—	—	—
1641		—	—	—	—	—	1:25	—	—	—
1006	2	—	—	—	1:25	—	1:25	—	—	+
1490		—	—	—	—	—	1:25	—	1:50	+
1161		—	—	—	1:25	—	1:25	—	—	—
1416	3	1:50	1:50	1:25	—	1:25	1:50	—	1:100	+
684		1:25	1:25	—	—	—	1:200	—	—	+
1071		—	—	—	—	—	—	—	—	—
1789	4	1:25	—	1:25	1:25	—	—	1:25	—	—
982		—	—	1:25	1:25	—	—	1:25	—	—
662		1:50	—	—	—	—	1:25	1:100	1:25	+
591	5	—	—	—	—	—	1:25	1:25	—	+
1565		1:50	1:25	—	—	—	1:100	—	—	—
1231		—	—	—	—	—	1:50	—	—	—
1628	6	1:50	—	1:25	1:50	—	1:800	—	1:100	+
1725		1:25	—	—	1:25	—	1:25	—	1:25	+
1640		1:25	1:25	1:25	—	—	—	—	1:50	—
1462	7	1:50	—	1:50	—	—	1:100	—	1:1600	+
1543		1:50	—	1:25	1:50	—	—	—	1:1600	—
1470		1:200	—	—	1:100	—	1:200	1:50	1:3200	+
1479	Controls	All remained negative								—
1230										

*Sera tested every three days after exposure. Birds killed on tenth day.

dilution of 1:200. *P. avicida* was recovered from 11 of the 21 birds at autopsy. The sera of the control birds remained negative when tested with all antigens.

Experiment II: In this experiment 21 birds were divided into seven groups of three birds each. (See table II.) A technic similar to that described above was used to inject intravenously each of six of these groups with a strain of the species of the genus Brucella, two strains of each of the three species being used. The birds showed a marked response in the production of agglutinins when injected with organisms of this genus. When tested with Brucella antigen, the blood sera of 2 birds reacted in a dilution of 1:800, eight sera reacted in a dilution of 1:1600, six in a dilution of 1:3200, and two in a dilution of 1:6400.

The results of using the seven *P. avicida* antigens on the blood sera of these birds showed a greater tendency to agglutinate

TABLE II—*Results of agglutination tests on sera of birds injected intravenously with species of the genus Brucella.*

Bird	Strain of Brucella Injected	Highest Titres Showing Complete Agglutination*								Brucella Isolated at Autopsy
		Standard Brucella Antigen	P. Avicida Antigens							
			1	2	3	4	5	6	7	
1716	*Br.*	1:1600	1:25	1:200	1:25	1:25	1:50	1:50	1:25	+
1556	*meliten-*	1:1600	1:25	1:50	1:25	1:25	1:25	1:25	1:50	+
1484	*sis* 42	1:800	1:50	1:50	1:25	—	1:50	1:25	—	—
1681	*Br.*	1:3200	1:25	1:100	1:25	1:25	1:25	1:50	1:50	+
1427	*meliten-*	1:800	1:25	1:25	1:25	—	—	1:25	1:25	—
1429	*sis* 58	1:1600	1:50	1:50	1:50	—	1:50	1:25	1:25	—
1424	*Br.*	1:1600	1:50	1:100	1:50	1:25	1:25	1:50	1:25	+
1776	*suis*	1:3200	1:50	1:200	1:100	1:25	1:50	1:100	1:50	+
1740	36	1:3200	1:50	1:200	1:100	—	1:100	1:50	1:25	—
1315	*Br.*	1:1600	1:25	1:50	1:50	1:25	1:50	1:25	1:50	+
1914	*suis*	1:1600	—	1:50	1:50	—	1:25	1:50	1:25	—
1410	35	1:6400	1:50	1:100	1:200	—	1:50	1:50	1:50	+
1612	*Br.*	1:3200	1:25	1:100	1:200	—	1:25	1:25	1:25	—
2739	*abortus*	1:6400	1:50	1:50	1:200	1:50	1:50	1:50	1:25	—
1623	31	1:3200	1:50	1:50	1:100	1:25	1:50	1:50	1:50	+
1508	*Br.*	1:1600	1:25	1:50	1:50	1:25	1:25	—	1:25	—
1262	*abortus*	1:3200	1:25	1:100	1:50	1:25	1:50	1:50	1:50	—
1314	32	1:1600	1:25	1:50	1:50	—	1:25	1:25	1:25	—
1319 1466 1515	Con- trols	All remained negative								—

*Sera tested every three days after exposure. Birds killed tenth day.

than did the use of similar antigens on blood sera obtained from birds injected with the various strains of *P. avicida*. Only 12 tests showed non-agglutination. Forty-eight tests showed a reaction in a dilution of 1:25, fifty in a dilution of 1:50, ten in a dilution of 1:100, and six in a dilution of 1:200. Brucella organisms were isolated from eight of the eighteen birds. All of the controls in this experiment remained negative when tested with all antigens.

Experiment III: Hughes and Pritchett[8] have shown that the normal portal of entry of *P. avicida* in the fowl is by way of the upper respiratory tract. Attempts were made to infect a group of birds by the implantation of various strains of *P. avicida* in

TABLE III—*Results of agglutination tests on sera of birds with intranasal implantations of P. Avicida.*

BIRD	STRAIN OF PASTEURELLA IMPLANTED	HIGHEST TITRES SHOWING COMPLETE AGGLUTINATION*								P. AVICIDA ISOLATED AT AUTOPSY
		STANDARD BRUCELLA ANTIGEN	P. AVICIDA ANTIGENS							
			1	2	3	4	5	6	7	
4217	1	—	1:25	—	—	—	—	—	—	—
4432		—	—	—	—	—	1:25	—	—	—
4068		—	—	—	—	—	—	—	—	—
4066	2	—	—	—	—	—	—	—	—	—
4097		—	—	—	—	—	—	—	—	—
4142		—	—	1:25	—	—	—	—	—	—
1459	3	—	—	—	—	—	—	—	—	—
1419		1:50	—	—	1:50	—	1:400	—	—	—
1414		1:25	—	—	—	—	1:50	—	—	+
1415	4	—	—	—	—	—	—	—	—	—
1418		—	—	—	—	—	—	—	—	+
1676		—	—	—	—	—	—	—	—	—
1708	5	—	—	—	—	—	—	—	—	—
1578		—	—	—	—	—	—	—	—	—
1483		—	—	—	—	—	—	—	—	—
1443	6	—	—	—	—	—	—	—	—	—
1744		—	—	—	1:25	—	—	—	—	—
1649		—	—	—	—	—	—	—	—	—
1554	7	—	—	—	—	—	—	—	—	—
1582		1:50	—	—	—	1:25	—	1:25	1:50	+
1749		—	—	—	—	—	—	—	—	—
1925	Con-	All remained negative								—
5672	trols									

*Sera tested 7, 14 and 21 days. Birds 1414, 1418, 1582 developed subacute cholera, killed on ninth day. Remainder killed 21 days.

the upper respiratory tract. Twenty-three adult birds were divided into seven groups of three birds each and one group of two birds. The latter served as controls, while a different strain of *P. avicida* was implanted intranasally in each of the remaining groups. Implantations were made from a bacterial suspension equal in density to McFarland's nephelometer tube 1 by means of a small cotton swab.

The results of agglutination tests using both *P. avicida* and Brucella antigens conducted upon the blood sera of these birds are shown in table III. Only ten of 147 tests with *P. avicida* antigens showed positive agglutination. Six samples showed agglutination in a dilution of 1:25, three in a dilution of 1:50, and one in a dilution of 1:400. When the blood sera of these birds were tested with Brucella antigen, only three sera showed a positive agglutination test; one reacted in a dilution of 1:25, while two reacted in a dilution of 1:50. The results show very conclusively that *P. avicida*, entering the body of the fowl through the natural portal of entry, does not call forth a marked response on the part of the host. *P. avicida* was isolated from the nasal passages of three birds when autopsied ten days after exposure to the organisms. All controls remained negative.

Experiment IV: Hughes and Pritchett[8] have shown also that *P. avicida* may produce localized infections, i. e., rhinitis, sinusitis, roup, and wattle involvement. An effort was made to study such cases, as it was thought that a chronic type of *P. avicida* infection might induce a greater agglutinin response on the part of the host. All available cases which showed a positive agglutination test for Brucella infection were studied as well as

TABLE IV—*Agglutination tests on sera of birds affected with localized P. avicida infection.*

BIRD	HIGHEST TITRES SHOWING COMPLETE AGGLUTINATION*								P. AVICIDA ISOLATED
	BRUCELLA ANTIGEN	P. AVICIDA ANTIGENS							
		1	2	3	4	5	6	7	
11535	1:50	—	1:25	—	—	1:50	—	—	+
685	1:25	—	—	—	—	1:25	—	1:50	+
1112	—	—	—	—	—	—	—	—	+
1113	—	—	—	—	—	—	—	—	+
1624	1:50	1:25	—	—	—	1:25	—	—	+
1815	1:25	—	—	1:25	—	—	—	—	—
1445	—	—	—	—	—	1:25	—	1:25	+
1406	1:25	—	1:25	—	—	—	—	—	—
16127	1:25	—	—	—	—	1:50	—	—	—

*Blood sera tested each week while under observation (6-8 weeks).

some cases which showed a negative test, but from which *P. avicida* was recovered from the area of localized infection. Both the direct plating method and the technic of Pritchett, Beaudette and Hughes[9] for isolating *P. avicida* from carriers of this organism were used.

The results of the agglutination tests by the use of *P. avicida* and Brucella antigens on the blood sera of these birds are shown in table IV. *P. avicida* was recovered from three birds whose blood sera failed to react to Brucella antigen. When tested with Brucella antigen the blood sera of four birds were positive in a dilution of 1:25, while two were positive in a dilution of 1:50. On the other hand, 52 tests made with *P. avicida* antigens were negative. Eight samples reacted in a dilution of 1:25, while the remaining three positive reactions occurred in a dilution of 1:50. All of these birds were under observation for four weeks and two were observed for eight weeks before being autopsied. The blood sera of these birds were tested once each week and the highest titre shown during the period of observation is recorded. There seemed to be very little change in the agglutination titres from week to week. The results of these agglutination tests would indicate that even in chronic, localized infections of *P. avicida* the agglutinin response in the fowl is not marked.

Pathology

All of the birds used in these experiments were autopsied and a histopathological study made of their various organs. *P. avicida* was recovered from 11 of the 21 birds into which this organism was injected intravenously. These birds were killed the tenth day following injection of *P. avicida*. As a rule they showed few macroscopic changes, congestion of the liver and gray foci on the surface of this organ being the only lesions noted.

Microscopic examination of tissues from these birds, however, showed marked and consistent lesions. Foci of perivascular hyperplasia of histiocytes were invariably present in the spleen about the smaller vessels, and occasionally similar areas were found around the larger vessels.

Marked hyperemia and both intra- and interlobular hemorrhage were the outstanding lesions in the lungs. Hyperplasia of histiocytes, both periatrial and perivascular, were of frequent occurrence. Similar foci often occurred in the submucosa of the large bronchioles. Pigment was present in considerable amounts in or near the areas of hyperplasia.

Advanced cloudy swelling was constantly present in the livers of all of these birds. In all cases the cytoplasm of the hepatic cells was completely broken down into rather coarse granules. A few scattered foci of hydropic degeneration were present in the liver of one bird (1462). The livers of three birds (1640, 1725, 1006) showed numerous small foci of fatty degeneration, while the liver of another bird (1416) showed advanced generalized fatty degeneration. Perivascular foci of hyperplasia occurred constantly, involving the interlobular vessels.

Cloudy swelling and necrosis of the tubular epithelium were the outstanding and constant lesions in the kidneys, the former predominating. Slight hyperemia and the presence of casts frequently occurred, while interlobular perivascular foci of hyperplasia occurred occasionally.

In the second group exposed to *P. avicida*, subacute cholera was produced in three birds (1414, 1418, 1582). These were infected by intranasal instillations of *P. avicida* in the upper respiratory passages. The organism was isolated from each of these three birds which were killed on the ninth day following exposure. The symptoms presented were paleness about the head, comb and wattles, and emaciation, which developed very rapidly. The macroscopic lesions noted in bird 1414 consisted of a slight general atrophy of the internal organs. Bird 1418 showed gray foci on the surface of the liver and a marked atrophy of the spleen. Bird 1582 showed gray foci on the liver, and ascites.

Four other birds, although they were apparently healthy, and bacteriological findings were negative when autopsied on the 21st day following exposure, may have shown evidence of previous *P. avicida* infection in that ruptured egg-yolks were found in two (1415, 1649), a third (1419) was an internal layer, while a fourth bird (1708) had a markedly atrophied spleen. No evidence was found to account for the lesions. All of the other birds gave negative bacteriological findings and showed no macroscopic lesions.

The histopathology of the organs of the three birds which developed subacute cholera was in general very similar to that previously described in birds exposed to *P. avicida* by intravenous injections. One bird (1414) showed small and numerous scattered foci of hydropic degeneration in the liver. In general, however, the lesions of these three birds were of greater intensity than those previously described.

Although the remainder of the birds in this group were apparently healthy and bacteriological findings negative, their organs showed microscopic lesions very similar in character to those shown in the injected birds. The livers of four birds showed numerous small foci of fatty degeneration, while the liver of a fifth bird showed generalized fatty degeneration.

The organs of the birds in experiment IV, aside from the localized lesions about the head, failed to show any macroscopic changes. When a histopathological study was made of the organs of these birds, however, it was found that all had microscopic lesions similar in character to those found in previous birds when dealing with *P. avicida* infection. The livers of these birds were particularly interesting in that they showed well advanced cloudy swelling, or what some authors might call extreme parenchymatous degeneration.

A macroscopic and microscopic study was made also of birds injected intravenously with Brucella organisms. The lesions produced were essentially similar in character to those which have been found to be quite typical of this infection in domestic birds.[2,3,10,11] An enlargement of the spleen is invariably observed in the early stages of Brucella infection. Later this organ atrophies. The liver may have gray foci and occasionally brown foci on the surface. This organ may also appear congested. In the later stages of the infection the liver becomes very friable. Aside from congestion of the kidneys, few macroscopic changes are noted in the remaining organs.

Microscopically the spleen shows perivascular foci of hyperplasia of histiocytes and congestion. Cloudy swelling occurs early in the hepatic cells and a general hydropic degeneration of these cells rapidly follows, with foci of necrosis and hemorrhage. Inter- and intralobular foci of hyperplasia of histiocytes occur early in the liver but tend to disappear as the parenchymatous tissue becomes hydropic. The tubular epithelium of the kidneys shows cloudy swelling and necrosis, the latter predominating. The glomeruli are usually hyperplastic. In the lungs periatrial and perivascular foci of hyperplasia of histiocytes are found. Such foci often appear in the submucosa of the bronchi and bronchioles. Congestion of the lungs is quite common.

Discussion

From the data presented it appears that there is a very marked response in the production of agglutinins when fowls are exposed

to the species of the genus Brucella and that there is a very poor response in the production of agglutinins when fowls are exposed to the various strains of *P. avicida*. Our findings in this respect are similar to those of Bushnell and Foltz,[12] who state that "the organisms (*P. avicida*) possess so little toxicity that they do not call forth any marked response on the part of the host." Beaudette[13] also has observed that avian blood sera in connection with *P. avicida* infection fail to show the presence of agglutinins to the extent that the agglutination test can be used with any degree of accuracy in the detection of infected birds. Pritchett, Beaudette and Hughes[9] have shown that fowls may be carriers of *P. avicida* infection. Beaudette[13] points out the fact that such "carriers" very rarely show the presence of agglutinins in their blood sera. Consequently we are forced to the conclusion that the agglutination test is of little value in detecting *P. avicida* infection in flocks of fowls. It has been our observation in these experiments and that of Huddleson and Emmel[2,3] that fowls exposed to Brucella infection rarely fail to develop specific agglutinins in their blood sera. Consequently the test for Brucella infection should have some diagnostic value when applied to a flock of fowls. These experiments show, as Mallmann[1] states, that there is cross-agglutination between *P. avicida* and the species of the genus Brucella. However, Mallmann's work was conducted on the sera of rabbits. There is no question but that various species of animals may differ in their ability to produce agglutinins when exposed to certain organisms and in the case of *P. avicida* infection it seems that this demonstrates the necessity of conducting such experiments on the natural host for the organism. It is possible, however, that one may be able to stimulate greater antibody production in the fowl by the injection of *P. avicida* when using the technic ordinarily used for this procedure and which Mallmann probably used in his investigation with the sera of rabbits. This procedure, however, would not be comparable to the stimulation of antibody production through natural channels.

It is interesting to note the variations in the agglutination tests when various strains of *P. avicida* were used as antigens. Beaudette[13] points out that the fluorescent type of *P. avicida* is extremely stable and difficult to agglutinate and that the blue type is more easily agglutinated and in some cases agglutinates spontaneously. Our results seem to bear out this statement in that antigens prepared from the typically fluorescent type

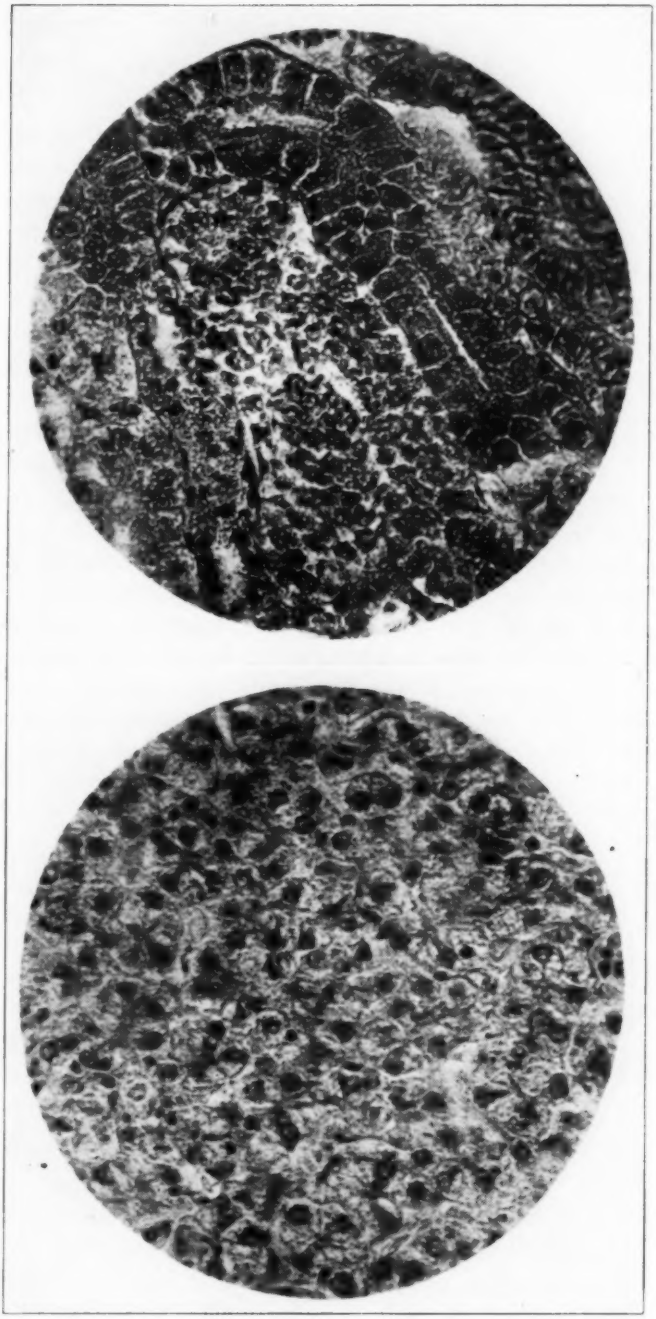

FIG. 1 (above). Section of kidney (bird 1418) showing degeneration of tubular epithelium; subacute cholera; exposed by intranasal instillation; *P. avicida* recovered at autopsy; blood-serum failed to react to both Pasteurella and Brucella antigens (x 500).
FIG. 2 (below). Section of liver (bird 685) showing parenchymatous degeneration of hepatic cells; localized infection (roup) with *P. avicida*; under observation eight weeks (x 500). (Photographs are by courtesy of Dr. L. H. Schwarte, Dept. of Veterinary Investigations, Iowa State Coll.)

agglutinated the sera of only two birds exposed to *P. avicida*, and gave a fewer number of positive tests than any other antigen used. On the other hand the typically blue type, when used as an antigen on the same sera, gave the greater number of positive tests, twenty-four. Other strains of *P. avicida* used as antigens were of the mixed type.

Brucella infection in the fowl produces a rather prolonged disease. Huddleson and Emmel[2,3] have shown it to extend over a period of 18 to 96 days, in cases where it proved a fatal infection. On this basis alone acute or even subacute cholera could be readily differentiated from Brucella infection. Sudden deaths as often seen in the acute type of fowl cholera have never been observed in Brucella infection. In the subacute type of fowl cholera, as observed in these experiments, emaciation developed very rapidly. In Brucella infection emaciation develops very slowly. *P. avicida* produces typically a septicemia or bacteremia. While the modus operandi is not exactly known as far as Brucella infection is concerned in the fowl, it is certainly not similar to *P. avicida*. There seems to be some difficulty in isolating Brucella organisms from fowls affected with Brucella disease. One has little difficulty in isolating *P. avicida* from fowls when dealing with this type of infection.

There is no abundant gross pathology in either *P. avicida* or Brucella infection. The spleen in the early stages of Brucella infection usually becomes enlarged; the liver becomes very friable, especially in the latter stages of the disease. While hemorrhagic enteritis is often observed in *P. avicida* infection, it is sometimes present in Brucella infection and is not uncommonly associated with considerable necrotic enteritis in the latter infection.

While there is some similarity in the histopathology of these two infections, one should be able to make a differentiation on this basis. The lungs and spleen present somewhat similar lesions. In the liver, however, Brucella infection produced general hydropic degeneration, often with focal necrosis and hemorrhage. *P. avicida* typically produced an extreme cloudy swelling or parenchymatous degeneration, occasionally with foci of fatty degeneration and rarely with foci of hydropic degeneration. The lesions found in the tubular epithelium of the kidneys are very similar. However, hyperplasia of the glomeruli occurs very commonly in Brucella infections and very rarely in *P. avicida*

infection. There is also more tendency to focal necrosis in the kidneys and liver in Brucella infection.

There should be no difficulty experienced in differentiating localized *P. avicida* infection, i. e., rhinitis, sinusitis, roup, and wattle involvement from Brucella infection, except possibly where one type of infection complicates the other.

Hughes and Pritchett[8] have observed that young birds which received intranasal instillations of *P. avicida* either died abruptly from typical fowl cholera or remained quite healthy. Beaudette[13] also has observed certain birds to be resistant to this infection. The data presented in our experiment in which fowls were exposed to *P. avicida* through the natural channels of infection confirm this opinion. It is quite interesting to note, however, that similar but not such extensive microscopic lesions were found in the birds which were apparently healthy as were found in birds affected with the subacute form of cholera, the birds being autopsied on the 21st day after exposure. As we were unable to isolate *P. avicida* from any of these birds which appeared healthy, we are of the opinion that the resistance of the birds overcame the infection shortly after exposure.

Huddleson and Emmel[3] have pointed out that Brucella infections in the fowl should be diagnosed only after a careful study of the history, course, symptoms, agglutination reactions, and gross and often microscopic pathology. Brucella infections in the fowl can be differentiated from *P. avicida*, as well as other infections, on this basis.

Summary

Experiments confirming the work of Mallmann on the interagglutinability of *P. avicida* and the species of the genus Brucella have been conducted. However, the fowl shows a marked response in the production of agglutinins when exposed to Brucella organisms but the reverse is true when exposed to *P. avicida* (seven strains). The agglutination test for Brucella would thus apparently have value in determining the presence of Brucella infection in a flock of fowls.

Acute or subacute fowl cholera can be differentiated from Brucella infection in the fowl bacteriologically, by distinct differences in the nature and course of the two infections, as well as by differences in the microscopic pathology produced by the causal organisms.

Localized *P. avicida* infection can be differentiated from Brucella infection by the macroscopic pathology produced about the head of birds by the former infection, *P. avicida* as a general rule being readily isolated from such lesions.

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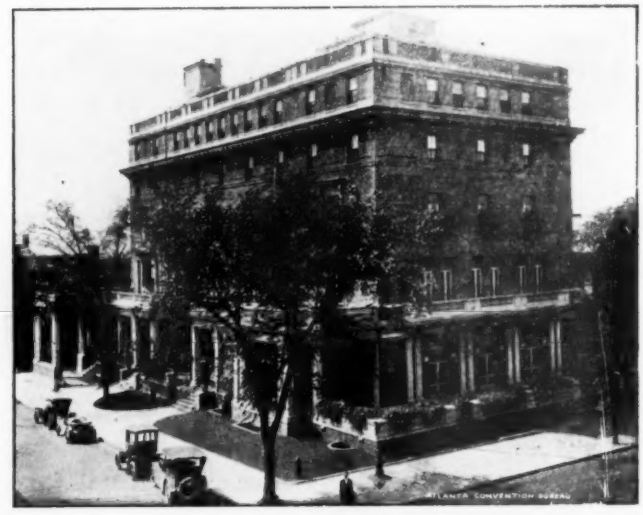

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A STUDY OF THE TUBERCULIN SENSITIZATION IN CATTLE SHOWING SUBCUTANEOUS LESIONS*

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For the past ten years, the veterinarians doing tuberculin testing of cattle in Montana have encountered in the dairy herds many animals which have reacted in some degree to the intradermic test, but in which no lesions have been demonstrated except the subcutaneous nodules which have been described as skin lesions. The frequent occurrence of these cases has constituted a rather serious problem for the man engaged in tuberculosis eradication work. Therefore, the laboratory of the Montana Livestock Sanitary Board has made pathological and bacteriological studies of lesions from many such cattle, and during the last four years an attempt has been made to throw some further light on the interpretation of the skin-lesion cases by making detailed observations on several herds where the skin lesions have been quite prevalent. It is the purpose of this paper to report the results of these observations. Before discussing this work, however, we shall briefly describe the general occurrence and distribution of the skin-lesion cases, and the nature of the lesions and reactions as found in our experience.

The subcutaneous nodules generally known as skin lesions have been recognized and described by a number of authors, and our observations of the nature of the lesions agree with the published descriptions. Apparently our experience is somewhat peculiar in that the majority of the lesions in our cases have been located on the teats.

The teat lesions occur in our experience only in dairy cattle. In our tuberculin testing, many beef herds and range cattle have been tested, but skin-lesion cases have rarely been encountered in these classes of cattle. The occurrence of these cases in dairy cattle may be still further limited to cows which are being milked, and to an occasional dairy bull, in which a

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skin lesion may be found on the scrotum. We have never observed subcutaneous lesions in heifers which have not yet been in the milking herd.

The teat lesions are evidently primary, due to a local infection, probably through an abrasion. The milk-duct is never involved, and lesions have not been found in the mammary gland. The teat lesion is often open on the surface of the skin, and ulcerated, showing a raw bleeding surface, with "punched-out" borders. These ulcers may heal, leaving a scar, with a hard subcutaneous nodule beneath. In some cases where teat lesions are present, there are subcutaneous nodules along the superficial lymph-channels over the udder, in the perineal region, and anterior to the udder. The skin lesions on other portions of the body have been on areas exposed to bruising, particularly the shoulders, where they come in contact with stanchions.

It is our observation that a majority of the cows showing subcutaneous lesions are sensitized to some extent to tuberculin and show more or less reaction to the intradermic injection of tuberculin. This sensitivity may be temporary and at a subsequent test the reaction may not develop. The reaction in the skin-lesion cases is usually not a typical tuberculosis reaction, although in some cases a reaction is obtained which is indistinguishable from a typical tuberculosis reaction. The atypical reactions are of such a nature that experienced operators feel that they can readily distinguish the type of reaction produced by a skin-lesion case from the reaction obtained in a case of internal tuberculosis. The swelling which develops is usually of small size, but it may be as large as many typical reactions. The distinction is made on the character of the swelling more than on the size. It is not firm and indurated, but is rather soft and indefinite in its extent. It is very evident that these atypical swellings are not the result of faulty technic, but are the result of sensitization to tuberculin, as this type of reaction is not encountered in testing range cattle, nor in young dairy cattle that have not yet been milked.

The problem of the interpretation of these reactions has been difficult to solve. It is our experience that these atypical reactions and skin lesions are most prevalent in herds in which no internal tuberculosis is occurring. Some of these herds have a past history of a heavy incidence of tuberculosis, while in others there is no history of the occurrence of tuberculosis. The question arises as to what significance these skin-lesion cases have with reference to tuberculosis eradication. There seems to be a pre-

ponderance of opinion at present to the effect that the skin lesions are not true tuberculosis and that the reaction is due to a group sensitization. If this be true, there would be no justification for destroying such cattle in a campaign to eradicate tuberculosis. If the organism involved be the tubercle bacillus, it is evidently attenuated, and may not be a factor in the spread of internal tuberculosis. There is also the question as to whether a veterinarian can certainly distinguish between a true tuberculosis reaction and the atypical reactions of skin-lesion cases, if the atypical reactors are not to be condemned as tuberculous.

For a number of years the Montana Livestock Sanitary Board laboratory worked on this problem from the laboratory standpoint. In 1924, we reported data on 60 cases of teat lesions in cows reacting to the tuberculin test. Since then we have studied 24 more teat-lesion cases and 14 cases of skin lesions on other parts of the body. Of the total of 98 cases, acid-fast bacilli have been found in smears in 73 instances. Of the 98 cases, 30 were studied histologically and, in 28 of these, the characteristic histopathology of tuberculosis was found. Animal inoculations were made with material from 48 cases, and 87 guinea pigs, 22 rabbits and 9 chickens were used. All of these were killed at intervals of from two to eight months after inoculation. In six guinea pigs and one rabbit, lesions were found in which acid-fast bacilli were demonstrated. Two of the guinea pigs, inoculated from one lesion, developed definite tubercular nodules, sections of which showed a histopathological picture indistinguishable from that of tuberculosis. All attempts at cultivation of the acid-fast bacilli on artificial media have failed.

As a result of the laboratory investigations, which are briefly summarized above, we feel that the question of the identity of the organisms involved has not been definitely settled, and that we are not yet justified in assuming that we are not dealing with the tubercle bacillus. It is certain that the organism involved is pathogenic to the extent of producing local lesions, and that histologically the lesion produced is identical with the tubercles of tuberculosis.

In an attempt to obtain further data bearing on this problem from the field standpoint, during the past four years we have run repeated combination tuberculin tests on several experimentally controlled herds in which skin lesions have been prevalent, and have made careful physical examination of all the cows in the herds. Our objects were to determine whether the subcutaneous

and ophthalmic tests would show sensitization corresponding to the intradermic results; to determine whether injections of avian tuberculin would indicate that we might be dealing with the avian type of the tubercle bacillus; and to check the results of leaving in the herds, under controlled conditions, the cattle that showed skin lesions and atypical reactions.

To avoid any misunderstanding, it should be stated at this point that it has always been the policy of the Montana Livestock Sanitary Board to condemn all cattle in which there was any reason to think that tuberculosis might be present, even though the reaction might not be typical. In control work, wherever acid-fast bacilli have been demonstrated, the diagnosis has been tuberculosis. It has been held preferable to destroy many no-lesion cases than to leave one tuberculous animal in a herd supplying milk to the public.

HERD 1

The principal object of the test on this herd was to obtain information as to the possibility of an avian source of the skin tuberculosis. The history of the herd was that, in the past, tuberculosis had existed to a considerable extent. For the two or three years previous to the present test, which was made in 1927, a number of reactors had been removed from the herd, but all of them were either no-lesion or teat-lesion cases.

In this test, 339 cattle were tested by the intradermic method. Of these, 255 received both bovine and avian tuberculin, the bovine being injected into the left caudal fold and the avian into the right. The results of the tests were as follows:

21 showed more or less reaction to one or both tuberculins.
7 of these 21 reacted strongly to the avian and not at all to the bovine tuberculin. One of the 7 showed a teat nodule.
5 reacted to the bovine and were negative to the avian tuberculin. Teat lesions were present in 3 of these 5.
6 reacted to both tuberculins. Teat lesions were present in 4.
3 of the reactors were tested with the bovine tuberculin alone, and teat lesions were present in all of them.

Of the cows reacting in some degree to the bovine tuberculin, only one reaction was considered typical, but the postmortem examination revealed no internal lesions. The cows which indicated sensitization to bovine tuberculin and which also showed subcutaneous lesions were killed and examined postmortem. No lesions other than skin or teat lesions were found on postmortem except a slight lesion in one mesenteric gland in a cow which also showed teat lesions. The relation between the mesenteric lesion and the teat lesions was not determined.

The results of this test do not indicate any relation between the teat lesions and sensitization to avian tuberculin. Of the seven cows reacting only to the avian tuberculin, only one showed a teat nodule, while of fourteen reacting to bovine tuberculin, ten showed teat lesions. Tuberculosis was found to exist in the poultry on one of the ranches, which may account for the sensitization of some of the cattle to avian tuberculin. It is of interest to note that on a retest made after ninety days, using both tuberculins, the seven cows which had reacted only to avian tuberculin showed no reaction to either tuberculin.

Herd 2

Between 1918 and 1927, this herd was tested eight times. In 1918, two reactors were removed on the subcutaneous test. No lesions were found on postmortem examination. In 1925, one animal was removed as a reactor, and only skin lesions were found. As a result of the test in the spring of 1927, five cows were classified as suspicious. On September 6, 1927, the five suspects were retested, with negative results. Another cow which failed to react was condemned on the laboratory report on skin nodules. This cow showed teat lesions with a large raw ulcer on one teat, several subcutaneous nodules on the udder, and a number of nodules on the flank anterior to the udder and in the perineal region above the udder.

On October 27, 1927, the entire herd was tested by a combination method. The total number tested was 209. All of them received bovine tuberculin in the left caudal fold and avian in the right. The 120 cows comprising the milking string were tested also by the ophthalmic method. The results follow:

16 animals showed more or less reaction to one or both tuberculins.
5 of the 16 reacted atypically to the bovine tuberculin only, and 4 of these showed skin lesions.
11 showed some intradermic reaction to both bovine and avian tuberculin.
No positive reactions to ophthalmic tuberculin were recorded.
Skin lesions were present in seven cases.

Three of the cattle which showed atypical reactions on this test were killed and postmortem examination made. All three had skin lesions, two showing teat nodules and the third a skin lesion on the shoulder. The cow showing the shoulder lesion had a lesion in the prescapular gland, and one of the teat-lesion cases also showed a lesion in the prescapular gland. No internal lesions were found.

It was observed that the water which the cattle drank ran through the poultry-yard before reaching the cattle. Therefore,

while there was no history of tuberculosis in the poultry, it was considered that there was a possibility that avian tubercle bacilli were present in the water. To check the existence of tuberculosis in the poultry, a postmortem examination was made of 100 mature chickens which were butchered three weeks after the cattle were tested. There were no lesions of tuberculosis in the chickens.

A combination test and physical examination was again made on this herd in December, 1930. Eighty-three cows of the milking string were tested by the subcutaneous, ophthalmic and intradermic methods. In the intradermic test, both bovine and avian tuberculins were used, the bovine being injected into the left caudal fold and the left labium of the vulva, and the avian tuberculin into the right caudal fold and the right labium of the vulva.

There were no reactions to the subcutaneous test. One cow showed a positive ophthalmic reaction. Sixteen cows showed slight reactions to one or both intradermic tuberculins. Eleven of these sixteen showed skin lesions. Two cows showed typical

TABLE I—*Test on herd 2, December, 1930.*

Cow	Lesions	Reactions					
		Intradermic				Oph.	Sub.
		Bovine		Avian			
		CF	V	CF	V		
1	Slight swelling, right shoulder	N	N	SA	N	N	N
2	None	SA	N	N	N	N	N
3	None	N	N	N	SA	N	N
4	Nodule above udder	SA	N	SA	N	N	N
5	None	SA	N	SA	N	N	N
6	None	N	N	N	N	P	N
7	Slight swelling, shoulder	N	N	SA	N	N	N
8	Swelling, right and left upper arms	SA	N	SA	N	N	N
9	Small nodule, left shoulder	N	SA	N	N	N	N
10	Teat nodule	A	N	SA	N	N	N
11	Teat nodule	N	N	N	N	N	N
12	Thickening, right shoulder	SA	N	SA	N	N	N
13	None	N	N	SA	N	N	N
14	Swelling, right arm and left shoulder	N	N	SA	SA	N	N
15	Teat nodule	N	N	N	N	N	N
16	Swelling, both arms and left shoulder	SA	N	SA	N	N	N
17	Swelling, both shoulders	SA	N	SA	N	N	N
18	Swelling, both shoulders	SA	N	SA	N	N	N
19	None	SA	N	SA	N	N	N

CF = caudal fold.
V = vulva.
Oph. = ophthalmic.
Sub. = subcutaneous.
N = negative.
SA = slight atypical.
A = atypical.
P = positive.

teat lesions without any tuberculin reaction. Sixty-four cows showed no reaction and no lesions. Table I shows the findings on the nineteen cows which showed either reaction or lesion.

HERD 3

Herds 3 and 4 have a history of tuberculosis. They have been tested for the past fifteen years, and much difficulty has been experienced in eradicating tuberculosis from these herds. In the past two years, three special tests have been run on these two herds in an attempt to determine whether any correlation existed between the reactions and the occurrence of subcutaneous nodules. The skin-lesion cases were left in the herds, and observations made as to the persistence of reactions and the possible development of internal tuberculosis in other animals as a result of leaving these cases in the herd.

The history of herd 3 showed that it passed two clean tests in May and October, 1925. In September, 1926, thirty-one reactors were removed from the herd, and also an old generalized case which had failed to react. In November, 1926, one reactor was removed, and in September, 1927, one cow reacted. In November, 1927, there were no reactors, but four reactors were removed in October, 1928.

The first test recorded here was made in October, 1929, and was a simple intradermic test. No typical reactions occurred, and no cattle were condemned. Nine out of thirty-one cows in the milking string showed teat lesions. Seven of these cases developed atypical tuberculin reactions. Three other cows had a history of having had sore teats, but showed no lesions at the time and no reaction.

In January, 1930, a combination test was run on this herd, the milking string at this time consisting of 28 cows. The same cows which reacted atypically to intradermic tuberculin on the first test again showed more or less sensitization. Two of the three cows mentioned above as having a history of sore teats had developed teat nodules but no reaction. Another cow had developed a slight teat nodule and a slight ophthalmic reaction, but no intradermic reaction. All the cows sensitized to the intradermic tuberculin showed a slight reaction to ophthalmic tuberculin, but none to the subcutaneous tuberculin. Cow 2, which had failed to react on the first test and which had a history of sore teats, developed a positive reaction to subcutaneous tuberculin. This animal was condemned, but no lesions were found on postmortem examination, except a slight teat nodule.

TABLE II—*Tests on herd 3.*

Cow	October, 1929		January, 1930					November, 1930					
				Reactions					Reactions				
				Intradermic					Intradermic				
									Bovine		Avian		
	Lesions	Intradermic Reaction	Lesions	CF	V	Oph.	Sub.	Lesions	CF	V	CF	V	Oph.
1	Teat nodules	A	Teat nodules	SA	N	S	N	Teat nodules	N	N	N	N	N
2	None*	N	None	N	N	N	P						
3	Many nodules. Teat and perineal region	A	Nodules on 3 teats and udder	A(P1)	N	X	N	Nodules, teat, udder and perineal region	A	N	N	N	N
4	Teat nodule	A	Teat nodule	A(P1)	N	X	N	Teat nodule	A	N	N	N	N
5	Teat nodule open	A	Teat nodule healed	A(P1)	N	S	N	Teat nodule	N	N	N	N	N
6	Teat nodule	N	Teat nodules open	N	N	N	N	Teat nodules	N	N	N	N	N
7	None*	N	Teat nodule	N	N	N	N	None	N	N	N	N	N
8	Teat nodules	A	Teat nodules	A(P2)	A(P2)	X	N	Teat nodules	N	N	N	N	N
9	Teat nodules	A	Teat nodules	A(P1)	N	X	N	Teat nodules open	N	N	N	N	N
10	None*	N	Teat nodule nodule on stifle	N	N	N	N						
11	Teat nodule	A	Teat nodule	SA	N	S	N	Teat nodule	N	N	N	N	N
12	No typical lesion	N	Slight nodule	N	N	X	N	None	N	N	N	N	N
13	Teat nodule	N	Teat nodule	N	N	N	N	None	N	N	N	N	N
14			None	N	N	N	N	Teat nodules	N	N	N	N	N
15								None	A	N	N	N	N

*History of a sore

CF = caudal fold.
V = vulva.

Oph. = ophthalmic.
Sub. = subcutaneous.
N = negative.
P1, P2 = size of swelling (B.A.I. code).

A = atypical.
S = slight.
SA = slight atypical.
X = positive but not extensive.

In November, 1930, another combination test was made on 37 animals, including the use of avian tuberculin. Four of the cows that reacted atypically to intradermic tuberculin on the other two tests had lost the reaction. Three of the cows showing teat nodules on the second tests, showed no lesions on the third. Cow 14, which showed nothing on the second test, had developed teat nodules but no reaction. Cow 15, which had been negative on previous tests, developed an atypical reaction to intradermic tuberculin but no lesions. The avian intradermic tuberculin and ophthalmic tuberculin produced no reactions in any animal.

Table II shows the results of the three tests, listing only the cattle which reacted or showed subcutaneous lesions.

Herd 4

The history of herd 4 showed that a few reactors had been removed each year for a number of years prior to 1929. This herd was tested in the same manner as herd 3. The results of the tests are shown in table III. On the first test 13 cows out of 33 in the milking string showed teat nodules. Eight of these cows showed atypical intradermic reactions. Three cows reacted which showed no lesions. One of these reactions was recorded as a typical reaction.

On the second test, three months later, 14 cows out of 46 in the milking herd showed teat lesions. At this time only four of the 14 showed any sensitization to intradermic tuberculin, while six of them showed slight ophthalmic reactions. There were two atypical intradermic reactions where no skin lesions were found. Three of the teat-lesion cases had developed since the last test, but only one of these three showed any reaction. Cow 5, which had teat lesions, gave a positive reaction to subcutaneous tuberculin, but was negative to the other tests. She was condemned, and no internal lesions of tuberculosis were found on postmortem examination. Acid-fast bacilli were found in a hyperemic mediastinal gland. Guinea pigs were inoculated with material from this gland with negative result. Cow 28 was condemned on a positive ophthalmic reaction. She showed teat nodules but no intradermic reaction. No internal lesions were found on postmortem examination.

On the third test, 17 cows out of 46 in the milking string showed teat nodules. Only six of these developed any intradermic reaction. Five other cows showed an atypical intradermic reaction but no teat lesions. Four slight reactions to avian

TABLE III—*Tests on herd 4.*

Cow	October, 1929		January, 1930					November, 1930					
				Reactions					Reactions				
				Intradermic					Intradermic				
									Bovine		Avian		
	Lesions	Intradermic Reaction	Lesions	CF	V	Oph.	Sub.	Lesions	CF	V	CF	V	Oph.
1	Teat nodule	A	Teat nodules open	N	N	S	N	Teat nodules	A	N	N	A	N
2	Teat nodule	A	Teat nodules	N	N	S	N	Small teat nodule	N	N	N	N	N
3	Teat nodule	N	Teat nodules	N	N	N	N						
4	None	A	None	A	N	X	N	None	N	N	N	N	N
5	Teat nodule	N	Teat nodule	N	N	N	P						
6	Teat nodule	N	Teat nodules	N	N	S	N	Teat nodule	N	N	N	N	N
7	Teat nodule	A	None	N	N	S	N	None	A	N	N	N	N
8	Teat nodule open	A	Teat nodules closed	N	N	X	N						
9	Teat nodule	A	Teat nodule	N	N	N	N	Teat nodule	N	N	SA	N	N
10	Teat nodule	A	Teat nodule	A(P1)	N	S	N						
11	None	T	None	SA	N	S	N	None	N	N	N	N	N
12	None	A	None	N	N	X	N	None	A	S	N	N	N
13	Teat nodule	N	Teat nodule	N	N	N	N	Teat nodules	N	N	N	N	N
14	Teat nodule	A	Teat nodule	A(P1)	N	X	N	Teat nodule	N	N	SA	N	N
15	Teat nodule	A	Teat nodules	SA	N	N	N	Teat nodules	A	N	N	N	N
16	Teat nodule	N											
17			Teat nodules	SA	N	N	N	Teat nodules	N	S	N	N	N
18			None	N	N	N	N	None	A	A	N	N	N
19			None	N	N	N	N	Teat	A	N	N	N	N
20			Teat nodules open	N	N	N	N	Teat nodule	N	N	N	N	N
21			None	N	N	N	N	Teat nodules	A	N	N	N	N
22			None	N	N	N	N	None	N	N	SA	N	N
23								None	N	N	N	A	N
24								Teat nodule	A	N	N	N	N
25								Teat nodule	N	N	N	N	N
26								None	A	N	N	N	N
27			Teat nodule open	N	N	N	N	Teat nodule	N	N	N	N	N
28			Teat nodules	N	N	XXX	N						
29								None	N	N	SA	N	N
30								None	A	N	N	N	N
31								Teat nodule	A	N	N	N	N
32								Teat nodule	N	N	N	N	N
33								Teat nodules	N	N	N	N	N

CF = caudal fold.
V = vulva.
Oph. = ophthalmic.
Sub. = subcutaneous.

N = negative
P1 = size of swelling (B.A.I. code).
A = atypical.
S = slight.

SA = slight atypical.
T = typical.
X, XXX = extent of reaction (B.A.I. code).

tuberculin were recorded, all of them in cases where no reaction to bovine tuberculin occurred, and two of them being in cows whoch showed teat nodules. There were no reactions to ophthalmic tuberculin.

Of the thirteen cows which showed teat nodules on the first test, five were no longer in the herd at the time of the third test, and one of those remaining showed no lesions. Four of the eight remaining in the herd had lost their sensitivity to tuberculin.

Table III shows the results of the three tests on this herd, listing only the cattle showing reactions or subcutaneous lesions.

Herd 5

This herd was chosen for study because, in contrast to herds 3 and 4, it has been free from tuberculosis since it was established about twenty years ago. In the last few years teat nodules have occurred in the cows to such an extent that they have interfered seriously with milking. A combination test and physical examination was made on this herd in November, 1930, at the same time as the last test on herds 3 and 4. The results of the test are shown in table IV.

Out of 17 animals in this herd, 9 showed teat nodules. All of these cattle developed a small atypical reaction to bovine intra-

Table IV—*Test on herd 5, November, 1930.*

Cow	Lesions	Reactions				
		Intradermic				Oph.
		Bovine		Avian		
		CF	V	CF	V	
1	Teat nodule, open	A	N	N	N	S
2	None	N	N	N	N	N
3	Teat nodule	A	N	A	N	N
4	None	SA	N	SA	N	VS
5	None	N	N	N	N	N
6	None	N	N	N	N	N
7	Teat nodule	SA	N	N	N	VS
8	None	SA	N	A	N	S
9	Teat nodule, slight	SA	N	SA	N	VS
10	Teat nodules	A	N	N	N	VS
11	Teat nodule	A	S	N	N	N
12	None	N	N	N	N	N
13	Teat nodules	A	N	N	N	S
14	None	SA	N	SA	N	N
15	None	N	N	N	N	N
16	Teat nodule	A	N	N	N	N
17	Teat slightly thickened	A	N	A	N	N

CF = caudal fold.
V = vulva.
Oph. = ophthalmic.
N = negative.
S = slight.
A = atypical.
SA = slight atypical.
VS = very slight.

dermic tuberculin, and three of them reacted slightly to avian tuberculin. In addition to the 9 cattle which showed teat lesions and atypical reactions, there were three which had no lesions, but showed slight sensitization to both bovine and avian tuberculins. Seven of the cattle that were sensitized to intradermic tuberculin showed a very slight reaction to the ophthalmic test.

Discussion

As stated in the first part of this paper, there were three principal objectives in running these field tests.

1. The first objective was to determine whether the subcutaneous and ophthalmic tests would show sensitization corresponding to the intradermic results. A study of the charts shows that we obtained no reactions to the subcutaneous test in the cattle which showed the atypical intradermic reactions under study. This fact apparently explains to some extent why skin-lesion cases did not present a problem in the early tuberculosis eradication work, when the subcutaneous method was the standard test.

It will be seen that on some of the combination tests no ophthalmic reactions were recorded, while on others very slight reaction was noted in part of the animals reacting to intradermic tuberculin. On the second test of herd 3, all the intradermic reactors were recorded as showing a slight reaction to the ophthalmic tuberculin. The ophthalmic reaction in these cases consisted of the production of a very small amount of a white, viscid exudate, with no apparent inflammation of the conjunctiva. In our routine testing these cases would have been considered negative.

A study of the tables brings out the fact that almost no reactions occurred as the result of the intradermic injection of tuberculin in the vulva. The upper and lower intradermic injections of bovine tuberculin were used in 257 tests. In 49 of these 257 tests, atypical reactions occurred in the caudal fold, while in only 6 cases did any degree of reaction develop in the vulva. This fact indicates a possibility that the lower injection might be an aid in differentiating between "skin-lesion" reactions and reactions indicating internal tuberculosis.

2. The second objective was to determine whether injections of avian tuberculin would produce reactions indicating that the subcutaneous lesions might be caused by local infection with the avian type of the tubercle bacillus. The only typical reac-

tions to avian tuberculin occurred in herd 1, where seven cows reacted strongly to avian tuberculin and not at all to bovine tuberculin. Only one of these animals had teat lesions. In the six tests in which both bovine and avian tuberculin were used, 68 atypical reactions to bovine tuberculin were recorded, out of a total of 647 animals tested. Of these, 32 cases also showed similar reaction to avian tuberculin. A total of 17 reactions to avian tuberculin were recorded in cows which were negative to bovine tuberculin. This number includes the seven strong reactions in herd 1 referred to above. These results would indicate that the cattle under study are sensitized slightly to both tuberculins, but more strongly to the bovine. There is no indication on the basis of tuberculin reactions that the subcutaneous lesions are caused by the avian type of tubercle bacillus.

3. The third object was to determine whether there was danger in leaving these skin-nodule cases in a herd, with reference to the possible development of internal tuberculosis. This was of particular interest in the herds which had a bad tuberculosis history. In the case of herd 1, all the cows which showed teat lesions were destroyed in 1927. Annual tests since that time had shown no reactors. In herds 3 and 4, all the teat-lesion cases were left in the herds after the test in October, 1929, and the last test was made in November, 1930. In that time no internal tuberculosis developed, as indicated by the fact that no typical reactions occurred.

The observations recorded in this report do not clear up the significance of the teat lesions and other subcutaneous lesions with reference to the work of tuberculosis eradication, but they bring out certain facts which may help in the solution of this problem, particularly from the standpoint of the field man doing the actual testing.

U. S. Tuberculosis Eradication Work in 1931

More cattle were tuberculin-tested in the United States during 1931 than in any previous year, according to a report, entitled "Sidelights on Tuberculosis Eradication," issued by the U. S. Bureau of Animal Industry. An average of more than one million tests were applied monthly during the year. More tests were applied in Wisconsin in 1931 than in any other state, the total being 1,608,144. The highest degree of infection, 12.5 per cent, was found to be in Rhode Island; the lowest, 0.6 per cent, in Alabama and South Carolina.

ENCEPHALITIS IN SHEEP*

By L. P. Doyle, *Lafayette, Indiana*
Veterinary Department, Purdue University
Agricultural Experiment Station

During the past several years, two instances of encephalitis in sheep have been encountered. In one instance one animal was affected in a flock of 45 head. In the other instance, five sheep in a flock of 35 died of encephalitis during a period of three months. During the following year, one lamb in a flock of 19 died, after showing symptoms of encephalitis, on the farm where the five animals had died the previous year. Aside from this one case, the disease has not recurred on these farms during a period of four years. The age of the animals affected varied from five months to two years.

The symptoms which were reported by the owners consisted of walking in circles and pushing against objects, such as walls, fences and feed-racks. One ewe which was observed at intervals for three days showed the following symptoms: At first she stood apart from the remainder of the flock and showed twitching of one side of the face. Later she lay on the sternum and rested the chin on the ground. At this time there was an abundant discharge of mucus from the left nostril, and twitching of both ears. When urged to rise, the ewe assumed a normal standing position of the hind limbs, but remained on her knees in front. Later she became comatose and showed marked flexion of the neck to the left.

Necropsy on this ewe showed marked icterus. The left maxillary sinus was filled with clear mucus; the other sinuses of the head appeared normal. Three botfly larvae were found in the frontal sinus. No other evidence of parasites in the head was found. There was some gross evidence of leptomeningitis,

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and apparently some increase in the quantity of brain fluid. Postmortem examination of the other affected sheep did not show any well-defined gross lesions.

Microscopic examination of the brain showed an apparently identical type of disease in the animals from the two farms. The only appreciable difference was a greater meningeal involvement shown by the ewe from the farm where only one animal was affected. This difference, however, was merely one of degree, as

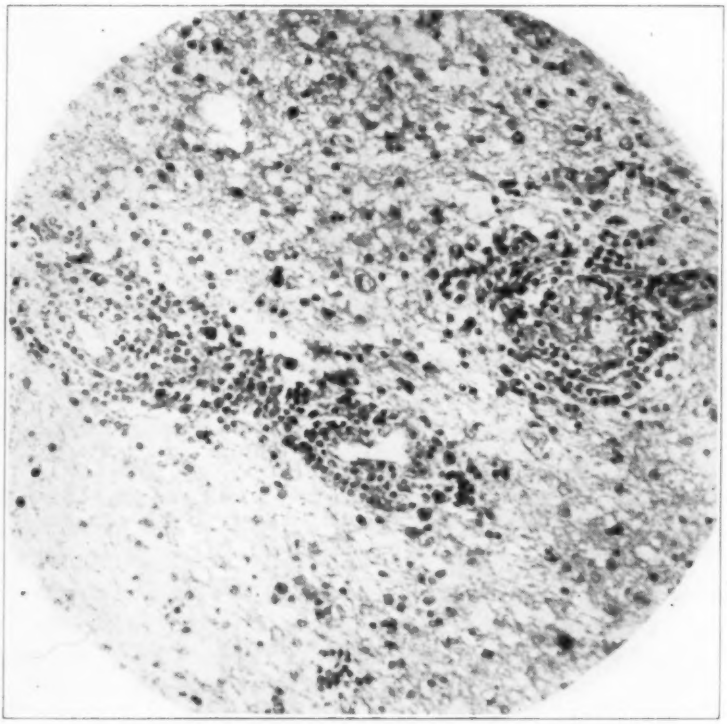

FIG. 1. A section of the medulla oblongata, showing well-marked "cuffing" of the blood-vessels.

all of the animals examined showed some inflammatory changes in the meninges of the brain. Well-marked microscopic changes were found in the cerebrum and in the brain stem.

The inflammatory change was characterized by perivascular accumulations of round or lymphoid cells. There were also areas of essential brain tissue which showed well-marked degenerative changes. These degenerated areas contained some large macrophagic cells. Among the round cells around the blood-vessels there

were a few neutrophilic polymorphonuclear leukocytes and an occasional large mononuclear cell. No cell inclusions were found in the nerve cells or in the inflammatory cells.

An interesting consideration in connection with these cases is the fact that symptomatically similar cases occur in which an encephalitis can not be demonstrated. This latter type of case probably results from intoxication or poisoning, while cases in which there is a well-defined inflammatory process in the brain very likely result from infection.

EPIDURAL ANESTHESIA IN THE EWE

By C. F. CLARK *and* L. B. SHOLL,

Michigan Agricultural Experiment Station,
East Lansing, Michigan

The above-mentioned method of producing anesthesia is well known to veterinarians and appears to be particularly applicable in cases of prolapsed uterus or vagina. The writers have not observed a report on the use of epidural anesthesia in the sheep. In the spring of 1932, the writers had occasion to treat cases of prolapsus vaginae in the ewe. Epidural anesthesia was tried and produced the desired effect rapidly and completely. The material used was a 2 per cent solution of dulcine in doses of 5 cc.

Technic: Locate the depression between the sacrum and the first coccygeal vertebra by alternately raising and lowering the tail. Insert a 16-gauge, 1½-inch needle in the center of this depression, tilting it slightly backward. When the needle encounters the floor of the neural canal, withdraw it slightly and inject the anesthetizing fluid slowly.

Books for the Veterinarian

A new catalog of books and periodicals dealing with veterinary science and animal husbandry has been issued by Bailliére, Tindall and Cox, of London. Both English and American publications are listed. The booklet is in three sections: (1) Veterinary Science; (2) Animal Husbandry (including breeding and management of small animals and poultry, beekeeping, etc.); (3) Periodicals (publications of direct interest to the veterinarian). Copies may be obtained from the publishers, 7 & 8, Henrietta St., Covent Garden, London, W. C. 2, England.

INFLUENCE OF LIGATION OF PANCREATIC DUCTS OF DOGS UPON SERUM AMYLASE CONCENTRATION. Carl E. Johnson and Carl H. Wies. Jour. Exp. Med., lv (1932), 4, p. 505.

In each instance a rise of several hundred per cent is observed in the amylase concentration of serum of the animals after an operation in which the pancreatic ducts were ligated, the high level being sustained for variable periods of several days followed by a decline to within at least 100 per cent of the preoperative level, whereas post-operatively, in the animals in which the pancreatic region was merely exposed, no such variation was observed. Sections taken from the pancreas for microscopic study showed atrophy of the acinar tissue with no obvious changes in the islands of Langerhans, in those animals whose ducts had been tied.

THE AGE FACTOR IN THE VELOCITY OF GROWTH OF FIBROBLASTS IN THE HEALING WOUND. Edward L. Howes and Samuel C. Harvey. Jour. Exp. Med., lv (1932), 4, p. 577.

The velocity curve of fibroplasia in the healing of wounds in young rats reached its end-point 13 days ahead of a similar curve for adults. Strength and fibroplasia were manifested one day sooner than in adults. The rate of fibroplasia during the accelerated phase was less in the young and it lasted longer. Retardation appeared later and was less in amount than in adult rats. Healing in the young therefore, is more rapid than in adults because fibroplasia begins earlier and is less retarded, and not because the rate of fibroplasia is greater. Growth of the young is not hindered by the process of wound healing.

RENAL THRESHOLDS FOR HEMOGLOBIN IN DOGS. I. DEPRESSION OF THRESHOLD DUE TO FREQUENT HEMOGLOBIN INJECTIONS AND RECOVERY DURING REST PERIODS. John A. Lichty, Jr., William H. Havill and George H. Whipple. Jour. Exp. Med., lv (1932), 4, p. 603.

"Renal threshold for hemoglobin" is used to indicate the smallest amount of hemoglobin which, given intravenously, will

affect the appearance of recognizable hemoglobin in the urine. The initial renal threshold level for dog hemoglobin is established by the methods employed at an average value of 155 mgs. of hemoglobin per kilo of body weight, with maximum values of 210 and minimum of 124. Repeated daily injections of hemoglobin will depress this initial renal threshold level on the average of 46 per cent, with maximum values of 110 and minimum of 60 mgs. of hemoglobin per kilo of body weight. This minimal or depression threshold is relatively constant if the injections are continued. Rest periods without injections cause a return of the renal threshold for hemoglobin toward the initial threshold levels. The authors believe that the minimal renal threshold level due to repeated hemoglobin injections is a little above the glomerular threshold which is assumed to be the base-line threshold for hemoglobin.

II. Renal Threshold for Hemoglobin in Dogs Influenced by Mercury Poisoning. William H. Havill, John A. Lichty, Jr., Gordon B. Taylor and George H. Whipple. Jour. Exp. Med., lv (1932), 4, p. 617.

The minimal renal threshold for dog hemoglobin is not modified by moderate doses of mercuric chlorid. This type of renal injury involves the epithelium of the convoluted tubules but the glomeruli escape. The evidence points towards the glomerular tufts as responsible for the passage of the hemoglobin from the blood plasma into the tubules. The glomerular tuft establishes the true hemoglobin threshold under these conditions. If the convoluted tubules are normal, it was noted that hemoglobin is taken into the epithelium and this explains the high initial renal threshold. With repeated injections this epithelium becomes stuffed with hemoglobin pigment fractions and can absorb no more, which explains the minimal or depression threshold. Further injury of this tubular epithelium with mercury produces no change in the minimal threshold unless actual destruction of the tubular epithelium occurs.

III. Tolerance for Mercury Poisoning Increased by Frequent Hemoglobin Injections. William H. Havill, John A. Lichty, Jr., and George H. Whipple. Jour. Exp. Med., lv (1932), 4, p. 627.

Frequent injections of super-threshold amounts of dog hemoglobin will cause deposits of pigment material in the renal

tubular epithelium. When this has happened, the dog will survive minimal lethal doses of mercuric chlorid with little evidence of renal injury. Some dogs tolerated twice the minimal lethal dose without severe reaction. There is no evidence that continued injections of dog hemoglobin in these amounts will cause injury or functional disability of the kidney. Rest periods will effect a disappearance of this pigment in the renal tubules.

IV. Hemoglobin Injections and Conservation of Pigment by Kidney, Liver and Spleen. The influence of diet and bleeding. William V. Newman and George H. Whipple. Jour. Exp. Med., lv (1932), 4, p. 637.

Anemia due to bleeding will accelerate the removal of pigment from the renal epithelium which was induced by the injection of hemoglobin. This would indicate a conservation of material by the kidney for use in construction of new hemoglobin. Pigment giving a positive stain for iron will be found in the liver and spleen when hemoglobin injections are given, regardless of the renal threshold. Removal of this pigment is accelerated by anemia due to bleeding and as a rule an anemic period of two months at a level of one-third normal will render the spleen, liver and kidney free from iron-staining pigment. Pigment giving a positive iron stain was frequently observed in the mesenteric and lower retroperitoneal lymph-glands. This is explained as a drainage of pigment, and phagocytes including pigment, from some organ in which the pigment deposit was primary. The authors conclude that the kidney is of considerable importance in the conservation of hemoglobin and hemoglobin split-products which are presumably utilized to build up new hemoglobin.

V. The Iron Content of Blood-Free Tissues and Viscera. Variations Due to Diet, Anemia, and Hemoglobin Injections. Robert P. Bogniard and George H. Whipple. Jour. Exp. Med., lv (1932), 4, p. 653.

The lowest iron content is observed in the pancreas, stomach, jejunum, colon and urinary bladder. These figures average from 1 to 2 mg. of iron per 100 gm. of fresh tissue. Smooth muscle and mucous membrane apparently contain little iron. Striated muscle (heart and psoas) shows a relatively low content of iron but uniform values close to 4 mg. per 100 gm. of tissue. Lungs show a considerable fluctuation with low iron values in anemia

(3.7 mg.) and higher in health (6 to 7 mg.). The spleen shows maximal fluctuations and the highest reserve storage of iron per 100 gm. of fresh tissue, 7 to 15 mg. in anemia, 25 to 50 mg. in controls, and 150 to 175 mg. when the animal is injected with hemoglobin. Bone-marrow of the rib runs parallel with the spleen. The iron content of the liver is depleted to 4 to 5 mg. per 100 gm. of fresh tissue in anemia, while there is an average of 25 mg. per 100 gm. of tissue in the normal dog. Injections of hemoglobin increase the level to 31 gm. of iron per 100 gm. The liver is considered the most active clearing-house for iron storage and utilization.

Coccidiosis in Gallinaceous Birds II. A Comparative Study of Species of Eimeria of the Chicken. Ernest Edward Tyzzer, Hans Theiler and E. Elizabeth Jones. Amer. Jour. Hyg., xv (1932), 2, p. 319.

Eimeria necatrix (Johnson 1930) produces a disease which is commonly fatal. The acute form, produced by heavy dosage of oöcysts, is characterized by intestinal stasis in association with hemorrhage and exudation into the small intestines, and death occurs from the fifth to the seventh day after infection. *E. necatrix* passes through two schizogonous cycles in the small intestines and completes its development usually in the ceca, to a slight extent in the large intestine, and in exceptional cases late stages may be found in the lower small intestine. Schizogony is continued through more generations than in *E. tenella* and the infection is more prolonged. *E. praecox* (Johnson 1930) is regarded as innocuous as far as direct injury to the tissue is concerned. Infection with this species is short-lived, immunity being promptly established. This organism is dispersed through the epithelium and shows no well-defined tendency to colony formation. Its development is very rapid. Similar exposure of chickens of various ages, including very young and others from 4½ to 10 weeks of age, to *E. necatrix* oöcysts, results in a markedly lower intensity of infection in the very young. This is explained on a physiological basis. There is no cross-immunity between *E. tenella* and *E. necatrix*. The egg-shell is regarded as a negligible source of infection to the baby chick. Long-contaminated surroundings are not a fertile source of infection. Subclinical cases of infection doubtless constitute a more important source of oöcysts than obviously ill birds. It is indicated that the continuance of occasional light infections in older previously exposed

birds may serve to carry the infection over from season to season, and it is quite probable that such constitute more or less intermittent sources of infection.

THE ANTIGENIC PROPERTIES OF RABIES VIRUS. Leon C. Havens and Catherine R. Mayfield. Jour. Inf. Dis., 1 (1932), 4, p. 367.

The antigenic character of rabies virus shown by these experiments does not strengthen the evidence of the identity of the Negri body with the virus of rabies. The Negri body probably bears the same relation to rabies as the Guarnieri bodies to vaccinia, whatever that relation may be. The author's work supports the view that viruses bear a greater resemblance to bacteria than they do to ferments and toxins, and also, that rabies thus resembles the viruses rather than the known protozoa. Specific flocculation of rabies virus occurs in appropriate dilutions of immune rabbit and guinea pig sera. Flocculation occurs with mixed virus and street virus. The serum of the rabies-immune guinea pig has been shown to possess specific complement-fixing antibodies for rabies virus. Immune rabbit serum is unsatisfactory for complement-fixation experiments with viruses, because of its anticomplementary nature.

FOOT-AND-MOUTH DISEASE LESIONS IN THE EAR EPIDERMIS OF INOCULATED GUINEA PIGS. Tom Hare. Jour. Path. and Bact., xxxv (1932), 2, p. 291.

The persistence of foot-and-mouth disease virus in the ear epidermis of guinea pigs, to at least the tenth day after infection, is associated with the presence of specific microscopic lesions to the eleventh day after infection. The formation of the initial lesions continues from the second to the fifth day as a characteristic degeneration of a single cell of the stratum germinativum. The formation of vacuoles and vesicles continues from the third to the seventh day and from the fifth to the eighth day, respectively. The vesicular exudate is discharged from the fifth to the eleventh day. The stages in the development of single lesions appear to occupy from three to six days.

THE NATURE OF THE ELEMENTARY BODIES IN PSITTACOSIS. S. P. Bedson, Jour. Exp. Path., xiii (1932), 1, p. 65.

Psittacosis virus can be thrown down almost completely by centrifugation at a speed of about 5000 r.p.m. The virus can

be freed from extraneous matter by fractional centrifugation and washing. The only particulate matter in a twice-washed deposit consists of minute bodies similar in every respect to those seen in preparations made from virulent material. The washed bodies are agglutinated specifically by an anti-psittacosis serum and fix complement in its presence, but do not react in either way with an anti-mouse-spleen serum. The author reaches the conclusion that the minute bodies are the virus.

FOOT-AND-MOUTH DISEASE IN VACCINE LYMPH. T. van Heelsbergen. Abst. Arch. Path., xiii (1932), 3, p. 514.

Norwegian vaccine lymph 980 produced foot-and-mouth disease, as well as the usual reaction to the vaccine. The possibility of dual properties in a single virus was ruled out by the successful separation and preparation of pure live virus strains, each producing the symptoms known to be associated with it and providing no cross-immunity.

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PERSONALS

Dr. John D. Groves (O. S. U. '13) has removed from Columbus to 92 E. Walnut St., Westerville, Ohio.

Dr. J. J. Van Voorhis (O. S. U. '15) has changed his address from Magnolia to Mineral City, Route 1, Ohio.

Dr. R. G. Elliott (Ont. '32) is practicing at Aberdeen, S. Dak., with his father, Dr. J. W. Elliott (Ont. '90).

Dr. James N. Mull (Ont. '32) is associated in practice with his father, Dr. A. A. Mull (Ind. '14), at Rushville, Ind.

Dr. A. W. Anderson (O. S. U. '26) is associated in practice with Dr. J. R. Robb (O. S. U. '28) at 880 Home Ave., Oak Park, Ill.

Dr. Carl J. Wallen (O. S. U. '23) has removed from Glendale to San Diego, Calif., where he is connected with the Poultry Pathological Laboratory of the California Department of Agriculture.

Dr. Lester J. Heiden (M. S. C. '23), of Escanaba, Mich., has been appointed Delta County Veterinarian by the Board of Supervisors and will make regular inspections of all dairy herds supplying raw milk in the county.

Dr. E. A. Rodier (Wash. '20), who has been connected with the Veterinary Research Laboratory at Pandacan, Manila, Philippine Islands, for a number of years, is now back in the United States. His present address is 202 Spaulding Street, Pullman, Wash.

ARMY VETERINARY SERVICE

Regular Army

The promotion of the following-named officers is announced:
Captain Herbert K. Moore to the grade of major.
2nd Lt. Arvo T. Thompson to the grade of 1st lieutenant.

Veterinary Reserve Corps

New Acceptances

Rice, Leon Henry.....Capt.....1612 2nd Ave., Kearney, Nebr.
Gloss, Ellis Harold.....2nd Lt....Alexandria, Minn.
Levy, Milton Charles...2nd Lt....795 Cole St., San Francisco, Calif.
Odom, Houston.....2nd Lt....P. O. Box 91, Auburn, Ala.

Promotions

To

Born, Arthur Leonidas..Capt.....Story City, Iowa
Pleuger, Carl August....1st Lt.....2146 Freeman Ave., Cincinnati, Ohio

ON TO ATLANTA

The Piedmont Driving Club, one of the oldest social clubs in the South, a place where many social celebrities have been entertained.

COMMENCEMENTS

ALABAMA POLYTECHNIC INSTITUTE

The commencement exercises of the Alabama Polytechnic Institute were held May 17, 1932. In the College of Veterinary Medicine the following graduates received the degree Doctor of Veterinary Medicine:

R. L. Durr
Howard Hayes
C. E. Kennedy
L. R. Mins
H. W. Sawyer
K. O. Smith
W. G. Sullivan
M. T. Thome

UNIVERSITÉ DE MONTRÉAL

Following final examinations on May 23, 1932, at the Ecole de Médecine Vétérinaire de la Province de Québec, Université de Montréal, the degree Doctor of Veterinary Medicine was conferred upon the following:

Lorenzo Brisson
Gaston Rodrigue

KANSAS STATE COLLEGE

The sixty-ninth annual commencement exercises of the Kansas State College were held at Manhattan, June 2, 1932. In the Division of Veterinary Medicine, the degree Doctor of Veterinary Medicine was conferred upon the following:

*Dalys Lewis Berry
*Loyd Edwin Boley
*Virgil Howard Clark
*Ben Harrison Dean
*Charles Eugene Dimon
David Franklin Engle
*John Lester George
*Harold P. Hartzell
*Melvin Eugene Hodgson
*Will Sydney Hornsby
*Chester Anson Paige
*Glen Frank Patton
Helen Sophie Richt
John Howard Rust
Frederick Ferdinand Schmidt
Fred Storz
Howard Irwin Thaller
*Arthur Frederick Van Meveren

Twelve of the graduating class (indicated by *) received commissions as second lieutenants in the Officers' Reserve Corps of the United States Army.

John Lester George was awarded high honors, and Loyd Edwin Boley was awarded honors in the Division of Veterinary Medicine.

Sophomore honors were awarded to Bradbury Bedell Coale and Carl William Schulz.

COLORADO AGRICULTURAL COLLEGE

Commencement exercises at the Colorado Agricultural College were held June 2, 1932. Twenty-seven graduates, including Miss Evelyn Hermann, daughter of Dr. A. A. Hermann, of Denver, received the degree Doctor of Veterinary Medicine. The graduates are:

William W. Aichelman
Haigaz H. Arshagouni
L. Duke Boston
John A. Clark
Heber H. Crowe
W. G. Duncan
Jean C. Flint
Geo. B. Fuller
Cecil L. Gates
John O. Goemmer
Curtis E. Hagler
H. Marvin Harvey
Floyd E. Heiser
Evelyn M. Hermann
John H. Jones
L. W. Jones
Al. B. Kight
Ed. E. Kraus
V. H. Magatagan
John R. Naylor
E. John Peters
William J. Purse
Irvin T. Reed
Herman J. Schick
Kenneth W. Smith
Walter H. Steele
William Kenneth Walker

The baccalaureate sermon, delivered on the Sunday before Commencement, was given by Dr. Mark A. Matthews, of the First Presbyterian Church, Seattle, Wash. Dean Harry M. Barrett, of the Educational Department, University of Colorado, delivered the commencement address.

A. AND M. COLLEGE OF TEXAS

At the commencement exercises of the A. and M. College of Texas, held June 4, 1932, the following graduates received the degree Doctor of Veterinary Medicine:

M. N. Bader
John M. Fitte
R. A. Goodman
S. E. Grove
E. A. Maier
Fred W. Pease

STATE COLLEGE OF WASHINGTON

Annual commencement exercises of the State College of Washington were held June 6, 1932. On this occasion the degrees Doctor of Veterinary Medicine and Bachelor of Science were conferred upon the following:

Percy Milton Aldrich
Elvin Walfred Almquist
Antonio Ancheta
O. Leighton Bailey
*John A. Baker
*John S. Bixby
Rolden F. Canfield
Ralph Wilson Case
Emil Edward Grinstead
Thomas Walter Jackson
George A. Morrison
Theodore Joseph Niemeyer
J. Dixon Nolan
*Clare W. Pritchard
*Mitchell J. Smith
John D. Winward

Four members of the class (indicated by *) were graduated with honors.

UNIVERSITY OF GEORGIA

The 132nd annual Commencement Day exercises of the State College of Agriculture and Mechanical Arts, University of Georgia, were held June 6, 1932. On this occasion the degree Doctor of Veterinary Medicine was conferred upon the following:

John D. Case, Jr.
Charles N. Cooper
Ancel L. Duckworth
Guy W. Eberhardt
Gordon L. Foy
Charlton J. Houston
Marcus B. Johnson
Melvin O. Nottingham
Shirley Shepard
George W. Shirley

Robert L. Willis

Charles N. Cooper and Guy W. Eberhardt were graduated with honors.

William D. Hiscock and Francis L. Tarver were granted the degree Doctor of Veterinary Medicine at the close of the 1931 summer session.

OHIO STATE UNIVERSITY

The fifty-fifth annual commencement exercises of the Ohio State University were held on June 13, 1932. The commencement address was delivered by Elmer Burritt Bryan, LL.D., L.H.D., president, Ohio University.

The College of Veterinary Medicine presented the following candidates for the degree Doctor of Veterinary Medicine:

Leo E. Andres
Edwin P. Barnes
Ray M. Batchelder
Morgan W. Bates
Lauren L. Bechtol
James R. Collier
George F. Delaplane
John R. Durigg
Guilford S. Elwood
Leo Fugate
Thomas W. Garrett
Lyle A. Gray
Carl W. Groppe
Russell E. Halstead
John M. Holmes
Robert J. Hoskins
John L. Jones
James R. Karr
John H. Knapp
Shefford S. Miller
Leroy Neuenschwander
Olof Norling-Christensen
Harvey F. Page
Arthur A. Rohrer
Elmer L. Rooks
Joseph R. Skala
Charles E. Streeter
Warren L. Tanner

Leslie A. Treat

The degree Master of Science in Veterinary Medicine was presented to the following candidates:

Howard M. Aitken
Theodore C. Fitzgerald
Amor E. Hancock
Leonard R. Richardson
Alan Cartwright Secord
Fritz Volkmar

Clifford C. Wagner

President George W. Rightmire conferred the degrees and, following the formal presentation of the candidates, the diplomas were awarded by Dean Oscar V. Brumley.

Nine members of the graduating class immediately filed applications for membership in the A. V. M. A.

MICHIGAN STATE COLLEGE

At the seventy-fourth annual commencement exercises of the Michigan State College, held June 13, 1932, the following graduates in the Division of Veterinary Science received the degree Doctor of Veterinary Medicine:

Frank R. Booth
Charles W. Huber
Clifford B. Line
Fred W. Meier
Kermit Schaaf
Oswar W. Schalm

J. Franklin Witter

Walter Johnson received the degree of Doctor of Veterinary Medicine at the conclusion of the summer session in 1931.

Four members of the 1932 class were graduated with high honors, and one with honors.

The Michigan State Veterinary Medical Association Prize of $25.00 was awarded to Oscar W. Schalm, whose senior record was the highest for the class.

IOWA STATE COLLEGE

Commencement exercises at Iowa State College were held June 13, 1932. On that occasion the degree Doctor of Veterinary Medicine was conferred on the following graduates:

Frank A. Anderson
Grant W. Anderson
Frank Blohm
George Buehler, Jr.
Bernard Buckley
Roy B. Conaway
Elwyn W. Coon
Thomas Dermody
Noran Ditman
Lester V. Dugan
Emery Enge
Greydon Forrest
Richard Geisler
Paul R. Granholm
Laurel Hade
Louis Heemstra
Cecil P. Hodson
Leonard Hoffmann
Maurice J. Johnson
Raymond W. Johnson
Willard Merchant
Clarence Pals
Eugene Peck
Alfred Peterson
Allen C. Peterson
Willson Reynolds
Wayne Riser
John Sanftner
Alfred Schladweiler
Ben E. Schoneman
Dwight A. Smith
Howard B. Stalnaker
Herbert Tabbut
Lloyd Thomsen
Harold E. Wicker
Julius Winkel

Telford W. Workman

Elwyn W. Coon led the group in scholarship and accordingly was awarded the George Judisch Prize, consisting of initiation fee and membership dues in the A. V. M. A. Dr. Coon was the honor man from the Veterinary Division and stood third in the list of twenty-eight for the entire College.

The G. G. Graham Prizes, awarded to outstanding students in clinical medicine on the basis of scholarship, attitude and adapti-

bility, were conferred as follows: First Prize, $15.00, to Maurice J. Johnson; Second Prize, $10.00, to Dwight Smith.

Graduates elected to the honor societies, Gamma Sigma Delta and Phi Kappa Phi, were Elwyn W. Coon, Paul R. Granholm and Telford W. Workman.

Undergraduates receiving honorable mention: Class of 1933, George L. Collins and Ben Meerdink; class of 1934, Edward E. Thompson and Weldon A. Winslade; class of 1935, George W. Mather.

Edward E. Thompson was the winner of the scholarship medal awarded October 21, 1931, by the Iowa State Student Chapter of the A. V. M. A., to the student with the highest scholarship record in the Division of Veterinary Medicine during the college year 1930-31.

UNIVERSITY OF PENNSYLVANIA

At the annual commencement exercises of the University of Pennsylvania, held June 22, 1932, the degree Doctor of Veterinary Medicine was conferred upon the following:

Mark W. Allam
Willard P. Boyer
John H. Brown
Arthur H. Craige
Roy F. Davenport
Richard L. Dolan
David T. Ensign
Darwin S. Fretz
John D. Gadd
Charles Hackenberg
Douglas G. Harrison
John W. Hillman
Jonathan K. Keim
Samuel V. Litten
Victor F. Mease
Raymond S. Moyer
Henry R. Recht
Sidney Seideman
Donald R. Skillen
Sidney W. Stiles
Albert E. Stockton
Edward E. Terry
Richard W. Tracey
John L. Wright
John T. Zurbrugg

The J. B. Lippincott Prize of $100.00, for the highest general average for the entire four years of the course, and the T. E. Munce Prize of $25.00, for the highest general average in the courses in Animal Husbandry, were awarded to Arthur H. Craige.

The Jeannette Blair Prize of $50.00, for the best work done in the Small-Animal Clinic, was awarded to Sidney Seideman.

The Leonard Pearson Prize of $50.00 was awarded to Donald R. Skillen. This prize is awarded to the member of the senior class who has shown, in the opinion of the veterinary faculty, by his scholarship, breadth of interest, personality and high character, combined with his ability to speak and write correct English, that he is most capable of dignifying and advancing veterinary science in research, in practice, in education and in its relation to civilization.

CORNELL UNIVERSITY

Annual commencement exercises at Cornell University were held June 20, 1932. The degree Doctor of Veterinary Medicine was conferred upon the following:

Raymond Ripley Allen
Martin David Baum
Samuel Berger
Curtis William Betzold
Morris Elmer Blostein
Walter Alan Boyd
Perry Thomas Combs
Joseph Raphael Conboy
Lewis Baxter Denton
Arthur Charles Eldred
William Morris Evans
John Joseph Farrell
Howard Kenneth Fuller
Louis Gaydosh
Ralph William Gifford
George Edward Gingras
James Henry Howard
Leonard Lawrence Howell
Hugh Willis Hunter
Burton Fuller Judson
Chester Jay Lange
Pincus Philip Levine
John McHool McCarthy
Harold Francis McDonald
Robert William Metzger
Henry Rohr Miller
Perry Sylvester Miller
John Carlton Minster
George Thatcher Parker
Charles Harry Payne
Niels William Pieper
Donald James Presler
Louis Charles Purmell
Albert Fred Ranney
Edward George Sadler, Jr.
Harold Louis Smead
Mark Sternfels
John Chester Stevenson
James Donald Sweet
David Augustine Walker
Edward Alexander White

The following prizes were awarded for the academic year 1931-32:

The Horace K. White Prizes (Meritorious Students):
First Prize ..... Pincus Philip Levine
Second Prize ..... John Chester Stevenson

The Jane Miller Prizes (Veterinary Physiology):
First Prize ..... Arthur Gordon Danks
Second Prize ..... Morris Harry Shapiro

The James Gordon Bennett Prize (Surgical Clinics):
Equally divided between ..... John Joseph Farrall and Hugh Willis Hunter

The Anne Besse Prize (Veterinary Medicine) ..... David Augustine Walker

The Charles Gross Bondy Prizes (Small-Animal Clinics):
First ..... James Henry Howard
Second Prize ..... Burton Fuller Judson

The Merry Prize (Anatomy):
Equally divided between ..... John Joseph Libra and Ralph Gordon Murch

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Eastern Iowa Practitioners' Clinic

The Eastern Iowa Veterinary Association has announced plans for a Practitioners' Clinic, to be held August 18, 1932, at the sale barn of Dr. F. M. Wilson, at Mechanicsville, Iowa. Dr. John B. Bryant, of Mount Vernon, is chairman of the committee in charge of arrangements.

MASSACHUSETTS VETERINARY ASSOCIATION

The annual meeting of the Massachusetts Veterinary Association was held in Boston, April 27, 1932. A banquet was served at the Hotel Westminster at 6:30, during which there was an enjoyable program of entertainment.

Before proceeding with the regular business of the meeting, the guest speaker, Dr. L. M. S. Miner, Dean of the Harvard Dental School, was called upon for an address. He spoke on diseases of the mouth and teeth, with special reference to Vincent's angina. Dr. Miner is an authority on this subject and has done a great deal of original research in connection with it. While he feels that possibly there is more evidence to prove that the fusiform bacillus, in combination with the *Spirochaeta vincenti*, should be considered as the primary etiological factor, he pointed out that there are other investigators who believe that this is secondary and that the underlying causes may be deficiencies, either of vitamins or of certain minerals, and that the organisms mentioned may be present as secondary or aggravating factors. Dr. Miner's address was extremely interesting and instructive throughout, and a great deal of valuable discussion followed. It was brought out, among other things, that many laboratory animals, including monkeys, had failed to develop the disease as a result of inoculation with scrappings from cases in the human. Dogs had not been used in the experiments, but it was very evident from the discussion that the disease is not at all uncommon in the dog. Demonstration of the organism is frequently made, and the symptoms, as they occur in the human, also are frequently observed.

Following Dr. Miner's address, the regular business of the annual meeting was taken up. Reports of various committees were presented, including one from a committee on rabies. This committee has been active in preparing a set of questions and answers on rabies for distribution to all dog-owners in Massa-

chusetts. The Association is publishing these in booklet form and has secured the approval of the State Division of Animal Industry, the State Department of Public Health, humane societies and other organizations. It is hoped that this booklet will do much to educate the public as to the nature of rabies and the proper methods for its control or elimination.

A pleasing incident of the meeting was a presentation to Dr. L. H Howard. The occasion was the fiftieth anniversary of his graduation. Dr. Harrie W. Peirce, in making the presentation, called attention to the fact that Dr. Howard, with a few other veterinarians, organized the Massachusetts Veterinary Association in 1884. Dr. Howard has been a prominent and active figure in veterinary matters for a great many years, and in his response to the presentation he related many things of interest about the pioneer days of the profession in Massachusetts. He ended by complimenting the Association on its present status.

The election of officers for the ensuing year resulted as follows: President, Dr. B. S. Killian, Somerville; first vice-president, Dr. F. G. Ruder, Amherst; second vice-president, Dr. S. T. Howland, Whitman; secretary-treasurer, Dr. H. W. Jakeman, Boston.

H. W. JAKEMAN, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held Wednesday, May 4, 1932, in the Academy of Medicine Building, 103rd St. and Fifth Ave., at 8:30 p. m.

Dr. O. E. McKim called the meeting to order, and called upon Dr. Robert S. MacKellar to introduce the well-known guest speaker, Dr. John R. Mohler, chief of the U. S. Bureau of Animal Industry. Dr. Mohler thanked the Association for the honorary membership which was conferred upon him last May, and gave an extremely interesting and instructive talk on the various activities of all branches of the Bureau.

Discussion was opened by Dr. Adolph Eichhorn. Dr. H. B. Leonard, of Albany, Dr. D. R. Gillies, of New York, in charge of meat inspection for the B. A. I. in this area, and the presidents of all of the local associations in the vicinity of New York,

joined in the discussion and paid their tribute of praise to Dr. Mohler as a scientist and as a man. The Association then extended a rising vote of thanks to Dr. Mohler.

The minutes of the previous meeting were read and approved. Case reports by Dr. H. K. Miller, of Mamaroneck, Dr. Jacob Lebish, of the Bronx, and Dr. Frederick W. Andrews, of Mount Kisco, were heard.

JOHN E. CRAWFORD, *Secretary.*

CONESTOGA VETERINARY CLUB

The Conestoga Veterinary Club held its nineteenth annual shad supper at the Stockyards Inn, Lancaster, Pa., May 19, 1932.

Dr. R. C. Gross, of Elizabethtown, presided. There were seventy members and friends present. Among the latter were: Hon. F. C. Musser, president of the Lancaster Live Stock Exchange; W. S. Hager, deputy secretary of the Pennsylvania Department of Agriculture; Dr. A. E. Wight, Washington, D. C.; Dean Geo. A. Dick, and Drs. C. J. Marshall, Wm. J. Lentz and John Beck, University of Pennsylvania; and others from the various veterinary organizations of eastern Pennsylvania.

Mr. Musser pledged the coöperation of the Live Stock Exchange with the regulatory authorities, and Mr. Hager discussed the new Pennsylvania regulations on Bang's disease. Dr. T. E. Munce stressed the growing importance of veterinary biologics, and outlined the progress made in eradicating tuberculosis from Pennsylvania herds. Dr. Wight spoke of the progress made throughout the United States in the work of eradicating bovine tuberculosis.

Dr. C. J. Marshall gave the principal address of the evening. His subject was "The Progress of Veterinary Medicine." During the course of his talk, he read excerpts from a book published in 1651, and from other books on the subject, published at intervals of about one hundred years. Other speakers included Dean Dick, Dr. M. F. Barnes, Dr. E. P. Althouse and Dr. U. S. G. Bieber.

The supper was a splendid one, and the club adjourned to meet with the Cumberland Valley Veterinary Club, at a joint meeting to be held at the State Laboratory, Harrisburg, Pa., on an invitation from Dr. Munce.

HENRY S. WEBER, *Secretary.*

NECROLOGY

LOUIS ARTHUR WILLSON

Dr. L. A. Willson died from an attack of angina pectoris at his home in Toronto, Ont., on April 3, 1932.

Born in Eglington, a suburb of Toronto, June 30, 1872, Dr. Willson received his education at the public and high schools in that city, and was graduated in veterinary science from the Ontario Veterinary College, in 1891, at the early age of nineteen. He engaged in private practice at Aurora, Ont., and in spite of his youth quickly gained the confidence of a large clientele.

In 1907, when the Federal Meat Inspection Service was inaugurated in Canada, he received an appointment in that service. One year later, he was promoted and placed in charge of this work in the city of Toronto, a position still held by him at the time of his death. He joined the A. V. M. A. in 1916.

Dr. Willson was the soul of honor and was very highly respected by those working under him, and his courteous manner at all times gained for him many loyal friends. He leaves to mourn his loss his widow, one daughter, and a host of friends.

G. H.

JAMES HENRY McCOY

Dr. James H. McCoy, of Bellingham, Wash., died at his home on March 27, 1932. He had been in poor health for over a year. He was a graduate of Washington State College, class of 1915, and for a time held the position of meat and milk inspector in the Health Department of Bellingham.

JAMES C. ELVIAGE

Dr. James C. Elviage, of Erie, Pa., died at his home, April 19, 1932, after an illness of several months. The true nature of his illness was not diagnosed satisfactorily, although it was known to be a cardiac affection.

Born near London, Ontario, October 7, 1862, he first learned to be a cheesemaker, later deciding to study veterinary medicine

Following his graduation from the Ontario Veterinary College, in 1890, he located at Sharon, Pa., where he practiced for five years. He then removed to Erie, where he built up an extensive practice. He retired in 1920 and since that time traveled widely, visiting nearly every state in the Union. Fifteen veterinarians attended the funeral services. Burial was at Lamberth, Ont.

E. E. B.

GRANT A. WEHR

Dr. Grant A. Wehr, of Denver, Pa., passed away April 29, 1932, at the age of 61. He was a graduate of the Ontario Veterinary College, class of 1896, and the McKillip Veterinary College, class of 1899. He joined the A. V. M. A. in 1927 and held membership in the Pennsylvania State Veterinary Medical Association and the Schuylkill Valley Veterinary Association.

PERCY LESTER ELLIS

Dr. Percy L. Ellis, of Merrill, Iowa, died at Sioux City, Iowa, May 13, 1932. His health began failing about a year ago, and he decided that it would be necessary for him to get away from the rigors of a country practice. He disposed of his practice and went to Columbus, Ohio, where he took up postgraduate work in the College of Veterinary Medicine, Ohio State University. While there, it was discovered that his health was permanently broken, and he decided to return to Iowa, where he would be among friends of long standing.

Born at Montour, Iowa, July 31, 1890, Dr. Ellis studied veterinary medicine at Iowa State College. As a student he was prominent in college activities and served as president of the Cardinal Guild, the student governing body of the institution. Upon his graduation in 1913, he entered practice at Merrill, where he remained for 18 years, except for the time spent in the service of his country during the World War.

Dr. Ellis entered the Veterinary Corps as a second lieutenant and was assigned to Camp Lee, Virginia. He spent 14 months in France and was discharged as a first lieutenant, at Camp Dodge, August 15, 1919. Following the war, he continued his interest in national defense and held a commission as captain in the Reserve Corps.

Although always a very busy man professionally, Dr. Ellis had time for civic activities. He served as president of the School

Board for four years, and as a member of the City Council for six years. He was Commander of the American Legion Post and trustee of the Methodist Church. He was a member of the Masonic Lodge of Le Mars and of the Abu Beker Shrine in Sioux City. He also belonged to the Knights of Pythias.

Dr. Ellis joined the A. V. M. A. in 1915. He was a member of the Iowa Veterinary Medical Association and served as president and secretary of the Inter-State Veterinary Medical Association. He is survived by his widow (née Marguerite Frances Huntley), two children, his father, a brother, and a sister.

CLEMENT H. PATTERSON

Dr. Clement H. Patterson, of Tiskilwa, Illinois, died at the Princeton (Ill.) Hospital, May 13, 1932, after a brief illness caused by a brain tumor.

Born at Tiskilwa, May 21, 1882, Dr. Patterson attended local schools. Following his graduation from Tiskilwa High School in 1910, he engaged in farming, with his parents, on their home farm. He entered military service in 1917, spent four months in training camps in this country and then was sent overseas as a sergeant in Field Remount Squadron No. 303. He served in this capacity until the end of the war and later with the Army of Occupation at Coblenz, Germany. He returned to the United States on May 21, 1919, and was honorably discharged June 21, 1919, after a short stay at the government hospital at Spartanburg, South Carolina, where he underwent treatment for a pulmonary trouble, resulting from having been gassed during his military activities in France. After farming for several years, he decided to study veterinary medicine and entered Iowa State College. Following his graduation in 1926, he entered practice at Tiskilwa.

Dr. Patterson joined the A. V. M. A. in 1928. He was a member of the Masonic order, Eastern Star, Modern Woodmen, and the American Legion. He is survived by his widow (née Bertha Dorr), his parents and an adopted son.

FLOYD E. WYANT

Dr. Floyd E. Wyant, of Albuquerque, N. Mex., died at his home, May 25, 1932, in his 46th year. Tuberculosis was the cause of death. Born near Colfax, Ind., Dr. Wyant was graduated from Thorntown (Ind.) High School in 1907 and studied

veterinary medicine at the Kansas City Veterinary College. Following his graduation in 1918, he entered the service of the U. S. Bureau of Animal Industry and was assigned to meat inspection at Chicago. Later he located at Thorntown, Ind., where he remained nine years, part of the time in general practice and the rest of the time as inspector at the Swine Breeders Serum Company plant. Ill health compelled him to go to New Mexico. He is survived by his widow (née Agnes Wood) and four small children.

JOHN W. LOCKWOOD

Dr. John W. Lockwood, of Fort Pierre, So. Dak., died May 13, 1932, at the age of 64. He was a native of Yorkshire, England, and came to the United States when only two years old. He studied veterinary medicine at the Chicago Veterinary College. Following his graduation in 1908, he located at Fort Pierre and remained there until his death. He is survived by three sisters and two brothers.

CHARLES E. MAGILL

Dr. Charles E. Magill, of Haddonfield, N. J., died at his home, May 22, 1932. He was born in Philadelphia, September 23, 1870, and was a graduate of the University of Pennsylvania, class of 1893. He had been engaged in general practice at Haddonfield since his graduation.

Dr. Magill joined the A. V. M. A. in 1927. He was a member of the Veterinary Medical Association of New Jersey. He is survived by his widow (née Virginia Hurff) and four children.

JOHN BRYCE

Dr. John Bryce, of Erie, Pa., the oldest living graduate of the Ontario Veterinary College, and the oldest graduate veterinarian in Pennsylvania, died June 9, 1932, after an illness of several months. A few years ago he suffered a paralytic stroke, and recently he was obliged to get around by means of a wheel chair. He maintained his interest in veterinary affairs, evidenced by his attendance at a recent meeting of the Northwestern Pennsylvania Veterinary Medical Club, as reported in the May issue of the JOURNAL.

Born at Mount Pleasant, Ontario, Canada, December 24, 1845, Dr. Bryce was graduated from the Ontario Veterinary Col-

lege in 1870. He located in Erie two years later and built up a very lucrative practice there. He always took a deep interest in saddle horses and was an honorary judge at many of the local horse shows.

Dr. Bryce's Masonic affiliations were numerous.

JACOB ORVILLE REED

Dr. J. O. Reed, of Allentown, Pa., died on June 10, 1932, as a result of an embolism that followed an injury to his leg while pushing his automobile.

Born at Danville, Pa., November 10, 1874, Dr. Reed attended local grade and high schools and Danville Academy before entering the Ontario Veterinary College. Following his graduation in 1896, he returned to Danville and practiced there until 1914, when he joined the field staff of the Pennsylvania Bureau of Animal Industry. At the time of his death he was in charge of the Allentown district for the Bureau.

Dr. Reed came into prominence in 1908 by being the first veterinarian to recognize foot-and-mouth disease in Pennsylvania, in the outbreak of 1908-09. He joined the A. V. M. A. in 1916 and was a member of the Pennsylvania State Veterinary Medical Association. He is survived by his widow (née Carrie Thompson) and two daughters.

T. E. M.

F. F. BROWN

Dr. F. F. Brown, one of the pioneer veterinarians in the Middle West, passed away at his home in Kansas City, Mo., June 11, 1932.

Born in Illinois, in 1862, Dr. Brown was educated in the public schools, Campbell University, Chicago Veterinary College and the Kansas City Veterinary College. Following his graduation from the Chicago Veterinary College in 1892, he practiced in Kearney County, Nebr., and in 1900 he became associated in practice with Drs. R. C. Moore and Sesco Stewart, and affiliated with the Kansas City Veterinary College.

Because of his character, educational attainment and recognition of the value of the practical application of knowledge, Dr. Brown was not only an unusual instructor but also an outstanding practitioner. He realized the value of an efficient veterinary service in the production of a wholesome milk supply

and was instrumental in the organization of the Kansas City Veterinary Association and the coördination of veterinary service and the activities of the Board of Health. He joined the A. V. M. A. in 1903.

That Dr. Brown was an efficient instructor will be attested to by the hundreds of veterinarians who were in his classes. As a practitioner, he was thorough and competent, and was a recognized authority on animal diseases in the Middle West. As a man, Dr. Brown was patient, conscientious and industrious, and his character was beyond reproach. As an associate in educational and professional service, he was a gentleman. His passing will be a distinct loss, not only to the veterinary profession but also to the live stock industry.

About forty veterinarians attended the funeral in Kansas City, and twenty were present at the burial in Hiawatha, Kan. Dr. Brown is survived by his widow, two sons and two brothers.

A. T. K.

J. W. OSBURN

Dr. J. W. Osburn, of Prescott, Arizona, died recently, the result of injuries received in an automobile accident. He was 74 years of age, had lived in Prescott for about 18 years, and was highly respected in that city. He was a registered, nongraduate practitioner.

PERSONALS

MARRIAGES

Dr. William Herbert Lowe (Col. V. C. '83-Amer. V. C. '88) to Mrs. Matilda K. Inglis, both of Paterson, N. J., June 15, 1932.

Dr. Taylor Prescott Rowe (U. P. '28) to Miss Ann Louise Elliott, both of Richmond, Va., June 18, 1932.

Elmer Walter Freytag to Miss Mary Louise Mayo, daughter of Dr. and Mrs. Nelson Slater Mayo, of Highland Park, Ill., June 8, 1932.

Dr. J. G. Horning (McK. '13) to Miss Ella Fahrenholt, both of Houston, Tex., June 4, 1932, at Lake Charles, La.

Dr. Hilan F. Keagy (Colo. '30), of Beverly Hills, Calif., to Dr. Evelyn M. Hermann (Colo. '32), of Denver, Colo., June 28, 1932, at Denver, Colo.

BIRTHS

To Dr. and Mrs. Edward A. Dornbusch, of Milbank, S. Dak., a son, Joseph Edward, May 12, 1932.

To Dr. and Mrs. Ray C. Ripple, of New Hampton, Iowa, a son, Robert Charles, May 16, 1932.

PERSONALS

Dr. A. L. Walsh (Chi. '20) has removed from Wykoff to Fairmont, Minn.

Dr. A. A. Fosterman (K. C. V. C. '18) has removed from Utica to Tyndall, S. D.

Dr. A. J. Osteen (Ga. '28) is Municipal Meat and Milk Inspector for Greensboro, N. Car.

Dr. H. J. Buehler (Mich. '29), formerly of Saint Edward, Neb., has removed to Sloan, Iowa.

Dr. D. Henry Wyatt (San. Fran. '15) has removed from Van Nuys, Calif., to Las Vegas, Nev.

Dr. E. G. Kerslake (Ont. '23), of Orono, Ontario, is now located in Toronto. Address: 500 Glenholme Ave.

Dr. G. S. Phalares (Mich. '30) reports a change of address from Chesaning, Mich., to 2839 Frederick Ave., Baltimore, Md.

Dr. John B. McQuown (O. S. U. '19) has removed from Globe to Tucson, Ariz., where he will engage in general practice.

Dr. D. K. Collins (O. S. U. '26), formerly of Rockland, Mass., has purchased the practice of Dr. J. A. Ford at Long Beach, Calif.

Dr. John H. Halton (San Fran. '03), who has been in New York City, gives a new address: 2020 San Jose Ave., Alameda, Calif.

Dr. H. W. Knoernschild (McK. '14) has requested a change of address from Sunbury, Pa., to 407 Seibert Court, West Lawn, Pa.

Dr. Henry B. Hannum (U. P. '20) has reported a change of address from Penbrook, Pa., to R. 3, Glen Moore Circle, Lancaster, Pa.

Dr. Charles B. Cain (Corn. '23), who has been at the Rockefeller Institute, Princeton, N. J., for some time, has returned to State College, Miss.

Dr. F. A. Young (Ont. '13), of Delphos, Ohio, sustained a fracture of a bone in his right hand, as the result of a kick from an equine patient recently.

Dr. G. C. Meskimen (Ind. '02), formerly of Vincennes, has located at Washington, Ind., for general practice. Dr. Meskimen was in Florida for several years.

Dr. J. E. Foster (Ont. '91), who has been in the life insurance business for about eight years, recently decided to resume private practice at Coshocton, Ohio.

Dr. Ralph A. Wilson (O. S. U. '30), formerly of Maplewood, has removed to 566 Valley Road, Upper Montclair, N. J., where he is conducting a small-animal hospital.

Dr. W. B. Chapman (St. Jos. '22), formerly of Leon, Iowa, has received an appointment in the U. S. Bureau of Animal Industry, and has been assigned to meat inspection at Boston, Mass.

Dr. F. E. Reddert (Colo. '28), formerly of Arboles, Colo., is now located in Santa Cruz, Calif., where he is engaged in the meat inspection service of the California Department of Agriculture.